

THE ASSESSMENT AND TREATMENT OF
CERVICAL DENTINAL SENSITIVITY

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ABSTRACT

This thesis is concerned with cervical dentinal sensitivity (CDS), with particular reference to its aetiology and treatment, together with an evaluation of the range of methods used to assess the condition with respect to patient response to treatment by desensitizing agents.

The original clinical study evaluated the comparative effectiveness of 2 strontium chloride hexahydrate (SCH)-containing dentifrices, similar except for their respective abrasive systems (silica-based and diatomaceous earth), in reducing cervical dentinal sensitivity (CDS). Both were equally effective, without any deleterious effect on plaque or gingivae.

Following cessation of 8-weeks' controlled use of both dentifrices, only a slight reversal of sensitivity levels, as assessed by tactile (Yeaple probe), thermal (cold air-dental unit syringe) stimuli, together with patient subjective response (Visual Analogue Scale VAS), was observed at 20 weeks, although overall, sensitivity levels remained significantly lower than at baseline. All the above methods of assessing pain from CDS appeared satisfactory.

On the basis of these findings a further portion of the investigation compared the various methods of evaluating patient subjective response (continuous 0-10 VAS, Numerical Rated Scale [NRS], Intensity [IVD] and Unpleasantness [UVD] Word Descriptors) following application of the above test stimuli used in both the 8 and 20 week studies. Both verbal and non-verbal techniques were able to quantify the sensory and affective aspects of CDS pain. However, the choice of word descriptor is important and care should be taken to use words which correspond to those generally used by patients when they describe the pain they feel.

Sequence of stimulus application is also important. This study demonstrated that patients perceived cold air from a dental unit syringe to cause the greatest discomfort and tactile the least, which appears to substantiate the sequence of stimulus application as used in the 8 and 20 week studies.

Comparison of recorded McGill Pain Questionnaires (MPQ) scores collected on 2 occasions from 40 patients (8-week study) demonstrated a very low percentage reproducibility (38% and 34.8% respectively from test and control groups), although words most commonly selected to describe pain appeared consistent with those in other studies.

The aim of the thermal probe studies was to evaluate the Biomat Thermal Probe (BTP) developed at this Institute as a potential aid to assessment of CDS pain.

The laboratory and clinical studies indicated that the BTP was accurate in in vitro measurement of temperature (range 0°C-59.9°C). Furthermore, compared with cold air from a dental unit syringe, the BTP provided an objective means of assessing patient response following thermal stimulation.

The BTP was also accurate in the measurement of cold threshold stimulation temperatures and, therefore, would be useful in clinical evaluation of desensitizing agents.

It was concluded that objective and reproducible methods of assessment are essential for accurate clinical evaluation of desensitizing agents. Tactile (Yeaple probe) and thermal (cold air and/or BTP) stimuli, together with patient subjective response using VAS go some way to meet such demanding criteria, while the Biomat Thermal Probe offers promise as a means of objective parametric assessment of CDS pain.

GLOSSARY OF ABBREVIATIONS

BIS-GMA	Bis(4-hydroxyphenol) dimethylmethane and Glycidyl Methacrylate
CDS	Cervical Dentinal Sensitivity
cm	Centimetre
EDTA	Ethylene Diamine Tetra Acetic acid
gm	Gram
HAD	Hospital, Anxiety and Depression Scale
INA	Intradental Nerve Activity
IVD	Intensity Verbal Descriptor Scale
kg/cm ³	Kilogram per cubic centimetre
Lp	Hydraulic conductance
ml	Millilitre (10 ⁻³ litre)
mm	Millimetre (10 ⁻³ metre)
mM	Milli molar solution
ms ⁻¹	Metres per second
MPQ	McGill Pain Questionnaire
NTG-GMA	N(p-Tolyl) Glycine and Glycidyl Methacrylate
PMDM	Pyromellitic Dianhydride and 2-hydroxyethyl Methacrylate
PNA	Pulpal Nerve Activity
ppm	Parts per million
p.s.i.	Pounds per square inch
RDA	Radioactive Dentine Abrasion value
rms	Root-Mean-Square
SCH	Strontium Chloride Hexahydrate
SEM	Scanning Electron Microscopy
SNA	Sensory Nerve Activity
µm	Micrometre
UVD	Unpleasantness Verbal Descriptor Scale
VAS	Visual Analogue Scale
VRS	Verbal Rating Scale

INTRODUCTION

Cervical Dentinal Sensitivity

Definition

Cervical Dentinal Sensitivity (CDS) has been defined as pain arising from exposed dentine, typically in response to chemical, thermal, tactile or osmotic stimuli, which cannot be explained as arising from other forms of dental defect or pathology (Addy et al.1985). The pain associated with CDS has been described as being rapid in onset, sharp in character and of short duration (Tarbet et al.1980, Trowbridge 1991), although, as Stephan (1937) and Chasens (1974) have indicated, the pain on occasions may persist as a dull or vague sensation in the affected tooth.

According to Dowell et al.(1985) correct diagnosis of the condition may be complicated by pain arising from the following clinical conditions:

- 1) Chipped teeth.
- 2) Fractured restorations.
- 3) Pulpal response to caries.
- 4) Pulpal response to restorative treatment.
- 5) The cracked tooth syndrome.
- 6) Palato-gingival groove.
- 7) Atypical odontalgia (Feinmann 1984)

Accurate assessment of the condition may also be difficult due to the subjective nature of the complaint (Everett et al.1966, Johnson et al. 1982).

The role of pulpal inflammation in the aetiology of CDS is somewhat controversial. Several investigators, on the basis of in vivo studies in animal and human teeth, have suggested that plaque products overlying exposed dentine may elicit an inflammatory response (Bergenholtz 1977, Bergenholtz et al.1982, Bergenholtz & Lindhe 1978,

Warfringe et al.1985). Recently several investigators (Kim 1990, Kim et al.1992, Olgart 1990) have suggested that repeated stimulation of sensitive teeth (in the animal model) may induce pulpal changes. Such changes could occur through induction of neurogenic inflammation and its subsequent effect(s) on pulpal blood flow. Presumably, if there is a decrease in pulpal blood flow, there would be a subsequent reduction in the outward fluid flow which may be insufficient to flush out any metabolites from the pulp or prevent the inward diffusion of potentially harmful plaque metabolites through open dentinal tubules from the oral environment. The relevance of such studies in the aetiology of CDS, however, may be questioned since teeth which appear to be sensitive to stimulation have little or no plaque present on the exposed dentine surface. Collaert and Fischer (1991) have also cautioned against extrapolating the results of these studies in coronal dentine (e.g., experimental cavity preparation and direct diffusion of plaque metabolites through to the pulp) to cervical dentine and the symptoms associated with CDS.

The term dentinal hypersensitivity has been used previously to describe this condition, which would imply excessive sensitivity or that the condition has a pathological basis. True hypersensitivity may represent a lowering of the sensory nerve excitability threshold (pain threshold) as proposed by Kim and co-workers (1990, 1992), but according to Trowbridge & Silver (1990), evidence to support such an hypothesis appears to be lacking.

The term cervical dentinal sensitivity, the term preferred in this thesis, would more accurately denote an exaggeration of a normal physiological response to the stimulation of freshly exposed dentine (Addy et al.1985, Trowbridge & Silver 1990). The prefix cervical indicates the location of the sensitivity, to distinguish it from that attributable to occlusal or approximal caries and/or its subsequent treatment.

Discomfort from CDS is a common finding within the adult population. Several investigators have reported that the prevalence of CDS ranges

from 8-35% of the population studied (Abel 1958, Jensen 1964, Graf & Galasse 1977, Kanapka 1982, 1990, Flynn et al.1985, Schaffner et al. 1988, Guo-Luo & Morimoto 1991, Fischer et al.1992). Graf & Galasse (1977) observed that one in seven patients (14.5%) presented with CDS due to gingival recession; whereas a British survey of 369 patients observed that approximately one in four patients (28%) claimed that they suffered from CDS, this figure, however, was reduced to 18% when the investigators examined the patients using a cold water mouthrinse (Flynn et al.1985). More recently, Fischer et al.(1992) reported that out of 635 patients, 157 (25%) reported having CDS, only 108 (17%) of these patients, however, were diagnosed as having CDS by these investigators using tactile and thermal stimulation. Schaffner et al. (1988) also reported that in 400 randomly selected patients from two age groups (20-30 years, 46-50 years), one in three suffered from CDS. A higher proportion of females appear to present with CDS (Orchardson & Collins 1984, 1987a, Flynn et al.1985, Fischer et al.1992) which may be due in part to better awareness in their appearance (Gesell et al. 1956) and associated improvement in oral hygiene (Buckley 1981).

Several investigators have provided information on the intra-oral distribution of sensitive teeth. The teeth most commonly affected are upper and lower canines and premolars (Graf & Galasse 1977, Orchardson & Collins 1984, 1987a, Flynn et al.1985, Addy et al.1987c, Schaffner et al.1988, Oyama & Matsumoto 1991). Sensitivity is usually associated with buccal or vestibular surfaces with exposed dentine (O'Leary et al. 1968, 1971, Woofter 1969, Sangnes 1976, Graf & Galasse 1977, Flynn et al.1985, Addy et al. 1987c, Orchardson & Collins 1987a), although other surfaces may be affected (Robb & Smith 1992). Exposure of buccal cervical dentine is probably due to excessive brushing (Rugg Gunn & MacGregor 1978, MacGregor & Rugg Gunn 1979). Although cervical dentine exposure increases with age, dentine sensitivity appears to peak in incidence at the end of the third decade and the beginning of the fourth (Franken 1931, Graf & Galasse 1977, Orchardson & Collins 1984, 1987a, Flynn et al.1985, Trowbridge 1991, Fischer et al.1992). This may

be due in part to age related changes in dentine and pulp (Flynn et al. 1985). Franken (1931) suggested that there was a seasonal incidence of CDS, with the condition being more severe in early spring and tending to decrease in the late summer or early autumn, although he provided no evidence to support this supposition. Extremes of temperature, heat or cold, appear to trigger sensitivity, cold being the more prevalent complaint (Kanapka & Colucci 1986, Ong & Strahan 1989, Trowbridge & Silver 1990).

Aetiology

According to Addy et al. (1985), once dentine is exposed to the oral environment it may be more prone to direct stimulation (e.g., cold, touch etc) and subsequent patient discomfort. The aetiology of denudation of the root surface by absence or loss of cementum and overlying periodontal tissues is multifactorial. According to Ong (1983) among the factors implicated are:-

1) Anatomical variation

- a) Chronic trauma from incorrect and/or over enthusiastic toothbrushing.
- b) Age: Gingival recession increases in severity with age.
- c) Chronic inflammatory periodontal disease.
- d) Malalignment of teeth.
- e) High fraenal attachment.

2) Dental procedures

- f) Defective restorations and dentures, for example, poorly contoured cervical restorations and inadequately designed denture clasps.
- g) Orthodontic trauma.
- h) Recession following periodontal procedures, eg root planing and surgery.

3) Other causes

- i) Habits, for example, fingernail stripping of the gingiva.
- j) Genetic predisposition.
- k) Occlusal trauma.

(Gorman 1967, Woofter 1969, Schluger et al. 1977, Carranza 1979, Grant et al. 1979)

Woofter (1969), Graf & Galasse (1977) and Schaffner et al. (1988) reported a correlation between gingival recession, wedge-shaped cervical lesions and oral hygiene habits, which suggested that abrasive toothbrushing may be responsible for CDS. There is evidence, however, that not all patients with gingival recession experience dentinal sensitivity (Flynn et al. 1985). At present there is no explanation for this apparent anomaly, although relevant factors may include age, rate of exposure of the dentine surface, the formation of secondary dentine and the effect of naturally occurring or other environmental desensitizing mechanisms (Addy et al. 1985).

Johnson et al. (1973), Brännström & Garberoglio (1980) observed that occlusion may have resulted from the formation of dead tracts with the laying down of reparative dentine, or from the development of sclerosed dentine within the dentinal tubules.

Occlusion of the tubule orifice may also occur as a result of smear layer and smear plug formation, which may include impacted dentifrice ingredients and oral debris (Hiatt & Johansen 1972, Pashley 1984, 1986a).

Several investigators (Brännström 1966, 1968, Ishikawa 1969, Addy et al. 1985, Absi et al. 1987, Yoshiyama et al. 1989, 1990, Oyama & Matsumoto 1991) have proposed that areas of sensitive cervical dentine display patent dentinal tubules. In other words there was a correlation between patent tubules and sensitivity. An SEM investigation by Absi et al. (1987) has also provided evidence (in extracted human teeth) that areas of dentine classified as 'hypersensitive' on the basis of clinical testing (probe, ethyl chloride and air-blast) showed a highly

significant increased number of open tubules per unit area (approximately 8x) compared to areas classified as 'non-sensitive'. Furthermore, the diameter of tubules in 'hypersensitive' areas were significantly wider (2x) compared to the diameter of tubules in 'non-sensitive' areas. These investigators, however, observed that open tubules (when present) were not uniformly distributed over the whole of the dentine surface, which appeared to correlate with the results of the methylene blue dye penetration studies in which the dye was restricted to a narrow zone of the exposed dentine. This observation appears to confirm clinical findings in which investigators have observed that not all exposed dentine is sensitive and response to certain stimuli (e.g., probing) is localised to a small area of dentine. There appears, however, to be no available evidence to suggest that 'hypersensitive' dentine is more permeable than 'non-sensitive' dentine. Närhi et al. (1992b), have also reported that in some human teeth, dentine hypersensitivity is not abolished even when the tubules are blocked, which suggests that other factors apart from tubule occlusion are involved in the prevention of stimuli transmission across dentine.

Effects of oral hygiene

Several investigators have suggested that plaque may play a role in the aetiology of CDS (Everett et al. 1966, Grant et al. 1972, Chasens 1974, Schluger et al. 1977, Carranza 1984). Other work, however, indicates that plaque is not a significant aetiological factor in CDS (Dowell et al. 1985), although several investigators stress the importance of good oral hygiene in its management (Grant et al. 1972, Chasens 1974, Schluger et al. 1977, Carranza 1984, Hovgaard et al. 1988).

Recent studies by Wallace & Bissada (1990) and Fischer et al. (1992) have suggested that CDS is positively correlated with previous periodontal therapy. Wallace & Bissada (1990) also claimed that there was an association between plaque accumulation after surgery and root sensitivity. Evidence from other studies, however, would appear to

strongly contradict such an assertion, since these studies support the concept that exposure of the root surface (gingival recession) and CDS are associated with excellent oral hygiene (Gorman 1967, O'Leary et al. 1968, 1971, Graf & Galasse 1977, Addy et al. 1987c, Addy et al. 1990b). L  e et al. (1978) also observed both loss of attachment and gingival recession and concluded that this reflected, not chronic inflammatory periodontal disease per se, but rather gingival recession due to toothbrushing. Although toothbrushing has been implicated as a major variable in exposing dentine, it is not the only factor which can abrade the root surface. Abrasive dentifrice components have also been implicated. Reisstein et al. (1978) observed by scanning electron microscopy that when brushed with a dentifrice, cementum was scratched more than by brushing with saline. The number of scratches increased with increased brushing time. There is also evidence that erosive agents, in particular dietary acids, are implicated (Peden 1977, Touyz 1983, Addy et al. 1985, 1987a,b, 1990a, 1991, Absi et al. 1985, 1987, Addy 1992). Davis & Winter (1980) demonstrated that enamel and dentine loss greatly increased when brushing is performed immediately after exposure of the surface to dietary acids compared with toothbrushing following exposure to water. More recently Addy et al. (1991) found that toothbrushing in vitro was unable to remove the acid labile smear layer, unlike certain dietary acids. Absi et al. (1992) also demonstrated that brushing in the presence of dietary acids appeared to accelerate the process of erosion and exposure of dentinal tubule orifices. Clark et al. (1985) reported a negative association between the frequency of acid food and beverage intake and persistence of CDS following a two month trial of a desensitizing dentifrice and a two week treatment of four applications of a topical fluoride varnish.

Clark et al. (1990a), in reviewing the available literature reported that no clinical evidence had been found to support the association between dietary acid and the persistence of CDS after treatment.

Problems associated with the assessment and treatment of CDS

Problems exist in the evaluation of the efficacy of a desensitizing dentifrice due in part to the lack of predictable, reliable and reproducible methodology for evaluating the subjective response of the patient, which can be further modified by social, cultural, psychological and situational factors (Ash 1986, McGrath 1986). Hence the variety of methods used to evaluate CDS, for example, mechanical and thermal stimuli and the patient's subjective assessment of pain in response to normal daily stimuli (Green *et al.* 1977, Minkov *et al.* 1975, Tarbet *et al.* 1979, 1980, 1982, Uchida *et al.* 1980). Opinions vary as to the reliability of the various methods of assessment (Green *et al.* 1977, Addy & Dowell 1983, Lecointre *et al.* 1986, Addy *et al.* 1987b). More recently efforts have been made to develop controlled reproducible stimuli more suited to the evaluation of CDS, for example the Yeaple probe, Yeh, Temptronic and thermo-electric devices.

In this thesis, the mechanism(s) of dentine sensitivity is discussed and the phenomenon of CDS described. The methods of assessing and treating CDS are critically appraised and related problems identified. Two strontium chloride hexahydrate-containing dentifrices (SCH), similar except for their respective abrasive systems, are compared in a two month randomised double-blind clinical study, involving 40 patients, to evaluate their comparative effectiveness in terms of CDS. The results of this study, together with the findings of a concomitant plaque and gingivitis study, and of a subsequent three month evaluation following cessation of dentifrice use, are presented and critically analysed. It is concluded that both SCH dentifrices were equally effective in reducing CDS. Following cessation of two months of controlled use of both dentifrices only a slight reversal of sensitivity levels was observed, although overall, sensitivity levels remained significantly lower than at baseline. Changing the abrasive component did not appear to affect efficacy of the SCH dentifrice(s). The two SCH dentifrices did not have any clinically significant effect

on plaque or gingival condition.

Recently a thermo-electric device (Biomat Thermal Probe) has been developed by E.H. Davies at the Institute of Dental Surgery for the purpose of evaluating the patient's subjective response to a thermal stimulus in clinical studies. The initial findings of in vitro and in vivo investigations are presented and the results critically analysed and compared with other methods of assessment.

To date, these results appear to be promising and would suggest that the Biomat Thermal Probe (BTP) is an objective and reproducible clinical tool and would be suitable for use in the assessment of CDS.

Initial findings of a series of in vivo studies designed to evaluate the subjective response of patients following application of test stimuli are also presented in this thesis.

CHAPTER 1

LITERATURE REVIEW

1.1. Innervation of Dentine

According to Närhi and co-workers (1990a,b, 1992a,b) electrophysiological recordings in experimental animals indicate that intradental A-type nerve fibres are responsible for the sensitivity of dentine and that the endings of the responding fibres are located in the pulp-dentine area. The exact mode of dentine sensitivity, however, is still unclear, although several hypotheses have been proposed. Currently, the most accepted mechanism of intradental nerve activation associated with CDS appears to be hydrodynamic in nature, although alternative mechanisms may be responsible (Horiuchi & Matthews 1973, Matthews 1977, Närhi *et al.* 1982b, Kim 1986a,b).

1.1.1. Neuroanatomy of Pulp and Dentine

The basic neuroanatomy of the dental pulp and dentine has been reviewed recently (Byers 1984, Johnsen 1985, 1990, Trowbridge 1986). Nerve fibres entering the teeth have been identified histologically as myelinated A-fibres and unmyelinated C-fibres (Trowbridge 1985). These fibres are grouped in bundles (Bernick 1948, Rapp *et al.* 1957) and enter through the apical foramina of the teeth, passing through the radicular to the coronal pulp where they fan out and diverge into smaller bundles (Gunji 1982, Dahl & Mjör 1973). Nerve divergence continues; individual A-fibres within small bundles lose their myelin sheath (Trowbridge 1986) and divide repeatedly before finally ramifying into a plexus of single axons known as the sub-odontoblastic plexus or plexus of Raschkow. The exact function of this plexus is unknown, as is the changing configuration of the plexus with dentine formation (Johnsen 1985). From this plexus nerve fibres are distributed towards the pulp-dentine border zone, with terminals showing a characteristic bead like structure (**Fig. 1.1.**).

Figure 1.1.

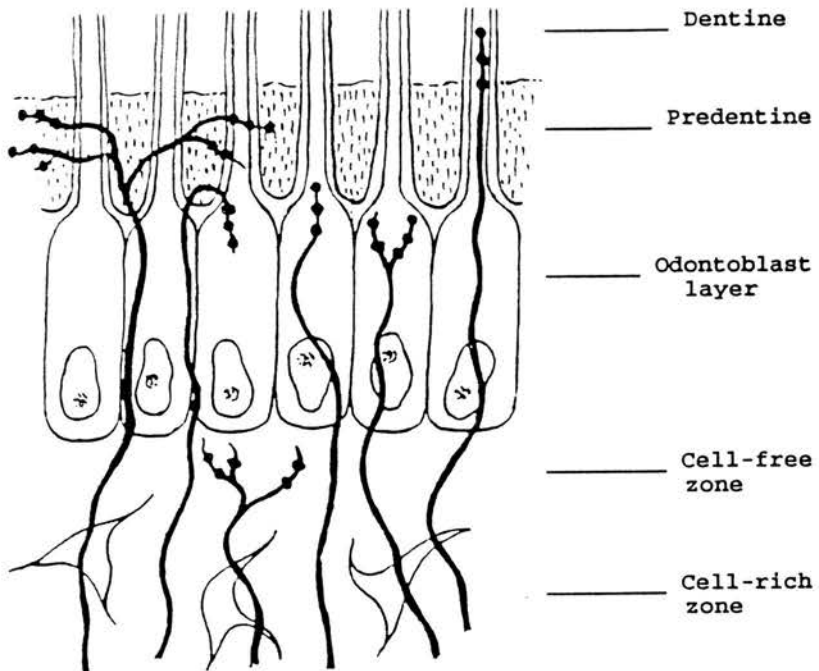


Diagram illustrating distribution of nerve fibres in the pulp dentine border zone.

Reproduced from: Review of Dental Pain: Histology and Physiology
Trowbridge, H.O. (1986) J. Endodont. 12:445-452.

Gunji (1982) has studied the distribution of nerve terminals arising from the sub-odontoblastic plexus in human molar teeth and classified four types of nerve endings, according to where they terminated. This has been summarised in the following manner by Trowbridge (1986).

a) Marginal fibres

These simple pulp fibres extend from the sub-odontoblastic nerve plexus to the odontoblast layer but do not reach the predentine.

b) Simple predentinal fibres

These fibres extend to the odontoblast/predentine border or enter the predentine. Gunji (1982) observed that some of these fibres ran straight or spiralled through a dentinal tubule along with an odontoblast process; others ran diagonally along the odontoblast/predentine border or within the predentine. Other fibres looped back towards the odontoblast layer.

c) Complex predentinal fibres

These fibres reach the predentine and undergo terminal ramification with multiple branches and multiple ending-like enlargements on each branch. The area covered by a single such terminal complex has been estimated to exceed $100,000\mu\text{m}^2$ in some instances. Penetration of this terminal type into dentine is limited to several μm .

d) Dentinal fibres

These fibres pass through the predentine without branching and enter the dentine through the dentinal tubule. The penetration is limited to approximately $100\mu\text{m}$.

Of the four types, the marginal fibres were the most numerous and the dentinal fibres the fewest (Trowbridge 1986).

Observations from early histological studies appeared to indicate that dentine was directly innervated (Mummery 1924, Wasserman 1939, Welling 1940, Bernick 1948, Powers 1952, Held & Baud 1955, Fearnhead 1963).

These observations, however, were open to conjecture. The use of silver impregnation techniques affected other structures such as reticular and collagen fibres as well as dentinal nerve fibres (Rapp et al. 1957). Bernick (1968) overcame this particular difficulty by the use of enzymes to digest collagen. Difficulties in interpretation also resulted from the limited resolution of the light microscope and from other, technical difficulties in the preparation of histological specimens. Later studies showed the presence of nerve-like fibres in dentine (Fearnhead 1957, Roane et al. 1973, Corpron & Avery 1973, Matthews & Holland 1975, Holland 1980). Fearnhead (1963) observed fine beaded fibres extending for a short distance into some but not all tubules. Penetration of fibres into dentine was limited to a few micrometres for most fibres, although some appeared to penetrate as far as 150-200µm (Byers & Kish 1976, Lilja 1979). Several investigators have suggested the presence of nerve structures in dentinal tubules, Frank (1968a,b) and Arwill (1967) using electron microscopy described such structures as terminal axons and sensory receptors. These observations were confirmed by nerve section studies (Arwill et al. 1973). More recently, sensory nerves were identified in dentinal tubules by autoradiography techniques (Byers & Kish 1976, Byers & Matthews 1981).

There is considerable variation in the number of dentinal nerve fibres from individual to individual and from tooth to tooth. Both Avery (1974) and Lilja (1979) demonstrated that only approximately 25-27% of the dentinal tubules (cuspal dentine) had associated nerve fibres. The coronal two thirds of dentine contained no neural structures in the dentinal tubules (Bernick 1948, Fearnhead 1957, Holland 1975, 1985, Lilja 1979). According to Lilja (1979) all the available evidence suggested that all neural structures observed were confined to the predentine and the most pulpal dentine, and, in the main dentine was largely devoid of nerve fibres. Lilja (1979) also found regional differences in the extent of dentine innervation.

Ten Cate et al. (1985) in commenting on the role of the odontoblast and

the extent of the odontoblast process within the dentinal tubule, suggested that the conflicting evidence provided by the differing technologies of the scanning and transmission electron microscopy may have accounted for the differences in observation and interpretation. They concluded that the odontoblast cell process extended only to the amelodentinal junction.

Holland (1990), however, stated that this position is still unclear. According to Holland, recent immunohistochemical findings and improvements in fixation techniques may have demonstrated structural evidence of odontoblastic processes in peripheral dentine. Szabo et al. (1985) have shown that a smooth lining, the lamina limitans, runs the whole length of the tubule. Other investigators (Sigal et al. 1984a,b, 1985, Aubin 1985), according to Holland, using polyclonal and monoclonal antibody labelling techniques have demonstrated the presence of components of the cytoskeleton in peripheral dentine. Several investigators (LaFleche et al. 1985, Frank & Steuer 1988) have suggested that under other forms of fixation the odontoblast process may have retracted (from its full length in the tubule) into the inner third of dentine. Holland (1975) has also observed (in the cat) that odontoblast processes vary in length and it is possible that at some sites the process may be as long as the dentinal tubule (Holland 1990). This supposition, if correct, could explain why some areas of exposed dentine are sensitive, while other areas are not (e.g., sensitive areas have tubules where the odontoblast process is closer to the exposed surface, whereas in non sensitive areas the process is confined to the inner one-third of the dentine).

Takahasi (1990), however, stated that evidence for an artifact of shrinking due to fixation was far from certain and doubted whether these new approaches to resolve the problem had made any more progress.

1.1.2. Classification of nerve fibres

Nerve fibres have been classified according to their conduction velocity and axon diameter (Lamb et al. 1980 after Erlanger & Gasser

1937) into A- ($A\alpha$, $A\beta$, $A\gamma$ and $A\delta$), B- and C- fibre types.

$A\alpha$ fibres have a diameter of 12-20 μ m and a conduction velocity of 70-120 m/sec. The primary function of these fibres is one of proprioception. $A\beta$ fibres have a diameter of 5-12 μ m, a conduction velocity of 30-70 m/sec, and are responsible for the transmission of touch and pressure, $A\gamma$ fibres have a diameter of 3-6 μ m and a conduction velocity of 15-30 m/s, and are responsible for motor function to the spinal nerves. $A\delta$ fibres have a diameter of 2-5 μ m with a conduction velocity of 12-30 m/sec and are responsible for the transmission of pain, temperature and touch. The second group of fibres, B-fibres have a diameter of 1-3 μ m with a conduction velocity of 3-15 m/sec and are responsible preganglionic autonomic function. The third group of fibres, C-fibres, are unmyelinated and have a diameter of 0.2-2 μ m with a conduction velocity of 0.5-2 m/sec. Their functions include postganglionic sympathetic, pain, and possibly heat, cold and pressure.

1.1.3. Neurophysiology of pulp and dentine

Dental pulp is innervated by both myelinated and unmyelinated fibres (Graf & Björclin 1951, Engström & Öhman 1960, Bueltmann *et al.* 1972, Beasley & Holland 1978, Johnsen & Johns 1978, Reader & Foreman 1981a,b, Byers 1984). By tooth eruption, both myelinated and unmyelinated nerves have reached the odontogenic regions and lie close to the odontoblasts (Fearnhead 1961, Avery 1971). Recent electrophysiological investigations on intradental nerves of experimental animals confirm histological evidence that two fibre groups (A- & C- fibres) exist, both fast and slow conducting, and that these groups are functionally different (Anderson *et al.* 1970, Närhi *et al.* 1982a,b, 1984, 1992a,b, Virtanen *et al.* 1983, Närhi 1985a,b, 1990a,b). There are also important differences in the quality of the pain evoked by A-fibres as compared with C-fibre stimulation (**Table 1.1.**).

According to Närhi (Närhi *et al.* 1982a,b, 1992a,b, Närhi 1985a,b, 1990a,b) it would appear that A-fibres are responsible for the

Table 1.1.

Characteristics of Sensory Nerve Fibres of the Dental Pulp

<u>Type of fibre</u>	<u>Myelination</u>	<u>Pain Characteristics</u>	<u>Stimulation Threshold</u>	<u>Location of Terminal</u>
A-delta	Yes	Sharp, pricking "fast" pain response	Relatively low	Pulp/dentine border zone
C-fibre	No	Dull, burning, aching, less bearable "slow" response	Relatively high	Distribution in the pulp uncertain

Reproduced from Mechanisms of Pain Induction in Hypersensitive Teeth Trowbridge, H.O. (1985)
(In Rowe, N.H. (ed): Proceedings of Symposium on Hypersensitive Teeth. Origin and Management.
University of Michigan, Pp 1-19).

sensitivity of dentine (dentinal pain). Most of the fibres fall into the category of A δ fibres (Trowbridge 1985), whereas C-fibres respond when external irritants (e.g., chemical agents) reach the pulp (pulpitis).

According to Närhi and co-workers there are other intradental nerve units which have conduction velocities above the range of the A δ fibres. These have been classified as A β fibres and appear to respond in the same way as A δ fibres to drilling, probing of dentine and air blast, which would indicate that both A δ and A β fibre units belong to the same function group. A β fibres may mediate non painful sensations induced by low intensity electrical stimulation of human teeth (Närhi 1990b).

1.2. Mechanisms of Stimulus Transmission across Dentine

Pashley and Parsons (1987) suggested that the mechanism of dentinal sensitivity transmission can be classified according to three main hypotheses:

- 1) Nerve endings or nociceptors that respond directly when the dentine is stimulated, these being located throughout the dentine.
- 2) Odontoblasts, being chemically or electrically related to nerves, function when depolarized as receptors generating nerve impulses.
- 3) Stimuli applied to dentine producing a displacement of dentinal tubule contents which could excite mechanosensitive nerve endings near the pulpal end of the tubules (hydrodynamic mechanism).

(Dowell & Addy 1983, Närhi 1985b, Byers 1984, Brännström & Åström 1972)

Several investigators (Horiuchi & Matthews 1973, Matthews 1977, Närhi *et al.* 1982b, Kim 1986a,b) have previously maintained that other mechanisms of pulpal sensory nerve activation may be responsible for the transmission of stimuli across dentine.

The work of Kim and co-workers (1985, 1986, 1987, 1989, 1991) also appears to support previous studies based on neurophysiological models that the net result of raising the intratubular K⁺ content is to render

the intradental nerves less excitable to further stimulation. Several investigators (Horiuchi & Matthews 1974 and Orchardson 1978) have also reported that chemical agents (e.g., 3M NaCl) did not elicit intradental nerve activity when applied in shallow dentinal cavities, but did cause excitation of A δ fibres when applied in deep cavities. The most probable mechanism for this mode of action being direct ionic diffusion (Kim 1990). According to Markowitz *et al.* (1991) the conclusions of these studies suggested that chemical agents were able to diffuse through the dentinal tubules and directly alter the extracellular fluid environment of the intradental nerves in the following manner.

- 1) By changing the extracellular fluid environment and altering the critical level for firing action potentials.
- 2) By directly altering membrane properties through a specific chemical interaction leading to a change in permeability.
- 3) By changing the microcirculation of the pulp.

More recently Kim (1990) has suggested that current experimental evidence supports two mechanisms for pulpal pain, namely the hydrodynamic and direct ionic diffusion theories

1.2.1. Direct stimulation of nerve fibres

Anderson *et al.* (1958) and Anderson & Naylor (1962) postulated that, if dentine was directly innervated, then chemical stimuli to the exposed sensitive dentine surface should cause pain. Application of algogenic (pain inducing) substances such as potassium chloride, acetylcholine, 5-hydroxytryptamine and histamine failed, however, to elicit a response; whereas when applied directly on exposed pulpal tissue an immediate response was elicited (Anderson & Naylor 1962, Brännström 1962, Anderson 1968, 1972). Similarly topical anaesthetic solution when applied to the exposed sensitive dentine did not decrease sensitivity (Anderson 1972).

On the basis of their findings, Anderson & Naylor (1962) proposed two

possible explanations:-

- 1) There were no nerve elements in dentine. When pain was evoked it was due to stimulation of receptor mechanisms in the pulp by a disturbance transmitted through the tubules by non-neural means.
- 2) There are receptor mechanisms in dentine which could be stimulated indirectly, but cannot be reached by direct stimulation from chemical agents because of some barrier to diffusion in the tubules.

Naylor (1963) observed that the very fast pain (cold) reaction times following thermal stimulation did, in fact, suggest the presence of a receptor located in dentine. Naylor (1968) later demonstrated that disruption of the odontoblast layer under a cavity did not block pain sensation following cold stimulation.

Application of sugar and calcium chloride solutions with high osmotic pressures did, however, produce pain in dentine (Anderson *et al.* 1958, Anderson & Ronning 1962), although these findings do not necessarily prove that a receptor mechanism is present in dentine, since there is evidence to suggest that nerves in the pulp may have been stimulated (Anderson *et al.* 1958).

Recent autoradiography studies of intradental nerves have demonstrated that nerve fibres in the pulp/dentine border area are injured by dentinal stimulation (Byers *et al.* 1987a,b, 1988), with a 50% reduction in the number of innervated dentinal tubules and in some instances loss of the nerve fibres in dentine. These results suggest that the existence of nerve fibres in dentine is not a necessary prerequisite for its sensitivity (Närhi 1990a,b), which also supports the evidence of Lilja (1979) that cervical and root dentine contains no intratubular nerves, but nevertheless is very sensitive. Several investigators (Byers & Taylor 1990, Byers 1992), however, have reported that following injury to dentine (rat molar) nerve fibres rapidly sprout under the injured cervical dentine provided the odontoblast layer is not destroyed. These sprouting calcitonin gene related peptide immunoreactive fibres (CGRP-IR) can temporarily innervate dentine of

the root. This effect, however, appears to diminish within 21 days. According to these investigators, it is also possible that exposed hypersensitive dentine may have an elevated number of nerve fibres in the underlying pulp and dentine. Although there are species and anatomical differences between rat and man, these are very interesting findings which could have implications (if proven) in determining the mechanism(s) of stimulus transmission across dentine in CDS.

Kramer (1955), also concluded that the lack of correlation between the incidence of disturbance of tubules contents and pain experience would seem to provide definite evidence that dentine sensitivity cannot be explained in terms of movement of tubule contents.

1.2.2. The Dentinal Receptor Mechanism Hypothesis

Proponents of the dentinal receptor mechanism hypothesis have suggested that the odontoblast has a special sensory function (although this receptor does not have to be the odontoblast), and that a functional complex with the terminal sensory nerve endings in close proximity to the odontoblast layer acts as an excitatory synapse (Frank 1963, 1968a,b, Arwill 1967, Dahl & Mjör 1973, Lilja 1979). Bernick (1948) was one of the earliest researchers to suggest such a relationship. These so called specialised junctional complexes (Frank 1963) were concluded to be a unique type of 'neurosensitive complex'. Arwill (1967, 1968) demonstrated the presence of cell projections in the pre-dentine, which he called associated cells, but not in dentine itself. The presence of tight junctions has also been described (Avery 1971, Scott 1974). Several investigators, however, have failed to establish the presence of any synaptic junction or special form of connection between odontoblast process and nerve endings (Fearnhead 1961, Anderson et al. 1970, Holland 1980), although an intimate contact between axon and odontoblast has been noted (Fearnhead 1963). Morphological evidence of a synaptic relationship between odontoblasts and sensory nerve endings, however, is lacking (Byers 1979, Gunji 1982).

Gunji (1982) hypothesised that free sensory nerve endings may in some way couple with the odontoblast process to form a mechanoreceptor complex capable of being stimulated when the odontoblast is mechanically deformed. This hypothesis, however, fails to explain why dentine continues to be sensitive following experimental destruction of the odontoblast layer (Brännström 1962, Brännström & Åström 1964, Ishikawa 1969, Hirvonen & Närhi 1986). Lundy & Stanley (1969) clearly demonstrated that although the odontoblasts had degenerated, clinical sensitivity was still evident. Transmission electron microscopy studies on dentine exposed to microbial products also confirm observations that dentinal sensitivity persists following the degeneration of both odontoblasts and intratubular nerve fibres located in the inner third of dentine (Lilja et al. 1982). These studies appear to contradict the hypothesis that the odontoblasts act as a dentinal receptor mechanism. Several investigators (Fearnhead 1967, Cadden et al. 1982, Holland 1985) have stated that it was unlikely that the odontoblast could perform the function of a special sensory receptor cell, while at the same time functioning as the specialised formative cell of dentine.

1.2.3. Evidence from histochemical and electrophysiological studies

Various histochemical and electrophysiological studies have investigated the possibility of a synaptic connection between terminal sensory nerve endings and odontoblast processes (**Transducer theory**). In order to substantiate this theory, the presence of a neurotransmitter substance, such as acetylcholine, would have to be demonstrated by evidence of acetylcholinesterase activity in the dentine (Rapp et al. 1968). Several studies support this hypothesis. Avery & Rapp (1959), Avery (1974) have shown that protoplasmic extensions of the odontoblasts were cholinesterase positive. Ten Cate & Shelton (1966) also demonstrated cholinesterase activity in both myelinated and non myelinated nerve fibres of the pulp, but not close to, or in odontoblasts or their processes. They concluded that if the transmission of impulses associated with dentinal sensitivity was via

a dentinal receptor mechanism, then there was no evidence to suggest that these impulses were mediated by cholinergic activity. There were, however, some technical problems in relation to the methods used by some of the earlier investigators, Ten Cate & Shelton (1966) used a more reliable and specific histochemical method.

Several investigators (Kukletová *et al.* 1968, Kroeger 1968, Turker 1975, Berman 1985, Kim *et al.* 1985, 1986) have suggested that nerve impulses in the pulp may be modulated by polypeptides, such as plasma kinins and Substance P (**Modulation theory**). Most studies, however, have failed to demonstrate any morphological evidence of a synaptic relationship between odontoblasts and sensory nerve endings (Byers 1979, Gunji 1982). Any direct effect of an external stimulus (e.g., thermal etc) on the pulpal nerves would also be unlikely due to the insulating properties of dentine (Phillips *et al.* 1956, Naylor 1963 Brännström & Johnson 1970, Phillips 1973) which, apart from the predentine and the most pulpal aspect is largely devoid of nerve fibres (Bernick 1948, Fearnhead 1957, Holland 1975, Lilja 1979). These findings would, therefore, lend support to an indirect stimulatory mechanism (Dowell & Addy 1983).

Several investigators (Scott & Tempel 1965, Scott & Stewart 1965, Scott 1966), however, have claimed that recorded electrical activity from dental nerve fibres in electrophysiological studies demonstrated the presence of receptors in dentine. Arwill *et al.* (1973) also reported that when electrophysiological recordings were made on teeth which had, had the inferior alveolar nerve resected, no impulse activity was recorded; whereas teeth on the control side with an intact nerve responded to locally applied stimuli. These investigators also observed associate cell degeneration on the nerve resected side. They postulated that the associate cell described in human teeth was actually a sensory neurone. This observation would appear to confirm the earlier electrophysiological studies of Scott and co-workers which gave some credence to the concept of a dentinal receptor mechanism. According to Anderson *et al.* (1970) cited by Matthews (1972), however, the evidence

from the studies of Scott and co-workers depended partly on their interpretation of the shapes of the recorded wavelengths as well as on the fact that these investigators were unable to record any activity until dentine was removed to within 100-200 μ m of the pulp where nerves fibres are known to be present. The possibility that the impulse activities recorded were those of pulp nerves, therefore, cannot be excluded. Matthews (1970) also reported that the response to stimulation recorded directly from an intact pulp was similar to that from the overlying dentine. Both Winter et al. (1963) and Matthews (1970) failed to demonstrate any recorded impulse activity which could be attributed to the odontoblast. Similarly, other studies noting the low membrane potential of the odontoblast also failed to demonstrate any recorded impulse activity (Kroeger et al. 1961, Winter et al. 1963, Matthews 1970). Horiuchi & Matthews (1971) also demonstrated that the recording system of Scott and co-workers could cause artifacts in the recorded activity. Furthermore, Horiuchi & Matthews (1974, 1975) observed that a recording electrode (Ag/AgCl) in contact with dentine may be capable of recording activity from beyond the immediate subadjacent tubules. Evidence from these studies, therefore, would indicate that the odontoblast does not possess the properties of a sensory receptor (Trowbridge 1982, 1985).

The exact mechanism of impulse transmission remains controversial. Mjör & Pindborg (1973) have stated that pulp and dentine sensation is characterised by being limited to pain only, irrespective of the initiating factor. According to Berman (1985), however, there is no direct support for any specialised terminal nerve receptors for hot, cold, electrical, osmotic, dehydration or chemical stimuli in dentine, although several investigators have demonstrated that, once the impulses reached the pulp, there were definite heat and cold sensitive nerves present (Matthews 1968, Scott 1974). Several investigators (Närhi et al. 1982a,b, 1992a,b, Hirvonen et al. 1984, Närhi & Hirvonen 1987) have shown that individual dental nerves (neurones) in animals respond to several different types of stimuli such as drilling,

probing, air drying and hyper-osmotic solutions. A study in humans also reported that cold perception was evident following application of a cold stimulus (Grüsser *et al.* 1982), although when these investigators anaesthetised the gingivae this relationship was reduced. Jyväsjärvi & Kniffki (1987) also reported that no sensation other than pain was perceived.

1.2.4. The Hydrodynamic Theory

Dentine is composed of hollow tubes containing a fluid or semi-fluid material (Neil 1850, Harriman 1872, Gysi 1900, Fish 1927, Kramer 1955). Neither Neil nor Kramer, however, were convinced that dentinal fluid movement was an acceptable explanation for the generation of pain (Rosenthal 1990). Gysi (1900) proposed that movement in dentinal canaliculi in either direction resulted in a sensation of pain. Fish (1927) had also proposed the idea of a fluid within the dentinal tubules, apparently extra-cellular (Coffrey *et al.* 1970). Ishikawa (1960) postulated that the pulpal lymph flow was continuous with that of the dentinal tubule fluid even though he failed to observe any pulpal lymphatics (in the dog). Stanley (1974) observed that free fluid made up about 2% of enamel volume and 25% of that of dentine. Kramer (1955) considered the dentinal tubule wall to be a relatively rigid structure, Johansen & Parks (1962) also observed that the walls were considerably more mineralised than the rest of the dentine. The diameter of these tubules was 2.5 μm at the pulpal end and 0.9 μm peripherally (Garberoglio & Brännström 1976). Brännström (1963a) reasoned that the conical shape of the dentinal tubules, together with the movement of fluid by capillary attraction, should obey the same physical laws as liquids in glass capillary tubes (Poiseuille's Law). The movement of fluid within the tubule was calculated to be about 2-4 micrometres per second (Berggren & Brännström 1965). Stanley (1974) also demonstrated that mobility of the fluid was high. Low hydrostatic pressures of 2 kg/cm^3 were also observed to elicit pain and to cause incremental flow of dentinal fluid towards the pulp as opposed to the

slow outward flow which normally appears to occur (Brännström 1966). This spontaneous rate of outward fluid movement, which flows down a hydrostatic pressure gradient from the pulp, is apparently too slow to activate mechanoreceptors (Pashley 1992). Johnson et al. (1973) also observed that in fractured dentine with exposed tubules, tubule contents could be emptied about ten times a day.

Brännström's observations from his experimental work on dentine sensitivity would suggest that the displacement of tubule contents, if rapid enough, could deform nerve fibres in pulp, predentine or damage odontoblast cells; both effects appear capable of producing pain (Brännström 1962). More recently, this definition has been refined to state that minute fluid shifts (either dentinal fluid or tubule contents) across dentine in either direction, in response to tactile, thermal or osmotic (chemical) stimuli, can stimulate mechanoreceptors in or near the pulp, which, in turn, excite sensory nerves to cause pain (Pashley 1985a,b, Pashley & Parsons 1987, Pashley 1992).

According to Pashley (1992) the hydrodynamic theory of dentine sensitivity as proposed by Brännström (1981) is based on the premise that sensitive dentine is permeable throughout the length of the tubules.

Currently, most investigators accept that dentine sensitivity is due to hydrodynamic fluid shifts which occur across exposed dentine with open tubules. This rapid fluid movement, in turn, activates the mechanoreceptor nerves of the A β and A δ class in the pulp (Pashley et al. 1992a)

1.2.5. Experimental evidence for the Hydrodynamic theory

In a series of experiments, Brännström and other investigators demonstrated that fluid shifts occurred through the dentinal tubules when pressure and dehydration procedures, as well as thermal stimuli, were applied to dentine (Brännström 1960a,b, 1962, 1963a,b, Brännström & Åström 1964, 1972, Brännström et al. 1967, Brännström & Johnson 1970, Haegerstam 1976).

1). The effect of pressure

The effect of pressure in teeth with cavity preparation made into dentine has been evaluated by Brännström (1963a,b, 1966). Following a decrease in pressure, an immediate pain response was elicited which persisted for as long as there was decreased pressure. Histological examination showed odontoblast nuclei in the tubules. Brännström concluded that these effects were probably due to intense evaporation from the dentinal surface (Brännström 1963a,b, 1966, Brännström & Åström 1972, Lilja et al. 1982). The dislocation is probably the result of the aspiration of the odontoblast into the dentinal tubules in connection with the capillary fluid flow (Närhi 1990a). Dislocated odontoblast nuclei have also been demonstrated in the dentinal tubules under stimulated dentine (Brännström 1963a,b, 1981, Hirvonen & Närhi 1986). Kramer (1955) observed this, but failed to correlate the incidence of disturbance and pain experienced. Brännström & Åström (1964), Lundy & Stanley (1969), Brännström (1981) and Hirvonen & Närhi (1986) have shown that dentine can still be sensitive even when the odontoblast layer has been destroyed.

Brännström & Åström (1964) postulated that rapid fluid shifts might activate nerves located at some distance from the tubules corresponding to the exposed dentine.

2). The effect of dehydration

Several investigators have demonstrated that the placement of dry absorbent paper on exposed dentine elicited a painful response, whereas with wet paper, no pain was experienced (Brännström & Åström 1964, 1972, Brännström 1966, Johnson & Brännström 1974). The scratching of the exposed dentine with a sharp probe or by dry chiselling also elicited a painful response. These procedures could cause the removal of dentinal fluid from the exposed dentine surface and by capillary action elicit an outward flow of tubule contents from the pulp (Brännström & Åström 1964, 1972, Brännström 1966, Johnson & Brännström

1974, Brännström & Johnson 1978), stimulating the odontoblast structure, causing pain. Recently Vongsavan & Matthews (1992a) have demonstrated that gentle probing caused inward movement of fluid.

Hypertonic solutions such as sugar and calcium chloride also elicit pain by the same effect of dehydration of the dentinal surface (Anderson et al. 1967a,b, Brännström & Åström 1972, Stanley 1974).

Bender (1978) has shown that the discomfort subsides when the irritant is diluted. This effect, too, can be explained by dentine tubule fluid movements, since fluids of a relatively low osmolarity (e.g., dentinal tubule fluid) will tend to flow towards solutions of higher osmolarity. When iso-osmotic solutions are applied no stimulus is perceived (Berman 1985). According to Haegerstam et al. (1975), the receptors of the tooth (in the cat) are not chemoreceptors, but probably mechano-receptors. This hypothesis is supported by the recording of nerve impulses following the application to dentine of stimuli known to create fluid movements in tubules (Haegerstam 1976).

Several investigators have also shown that intradental A- nerve fibre units in animals respond to several different types of stimulus affecting dentine such as drilling, probing, air drying and hyper-osmotic solutions (Närhi et al. 1982a,b, 1992a,b, Panopoulos 1983, Hirvonen et al. 1984, Närhi & Hirvonen 1987). These stimuli induced pain when applied to human dentine (Anderson 1963, Brännström 1963a,b, 1966, 1981). Lilja (1980) demonstrated sensory differences between crown and root dentine using dry absorbent paper, air blast and calcium chloride solution. Pain in crown dentine was sharp and shooting, that in root dentine, dull and often of longer duration. Acid etch treatment has also been utilised in studies of dentine sensitivity. This treatment is known to remove any drilling debris contributing to the smear layer and to open the dentinal tubules (Brännström 1979). Acid etching may also enable the stimulus to evoke intratubular fluid movements, which in turn makes the intradental nerve units more responsive (Närhi et al. 1982a,b, Panopoulos 1983). This factor may not have been accounted for in studies which failed to demonstrate a response to osmotic stimuli

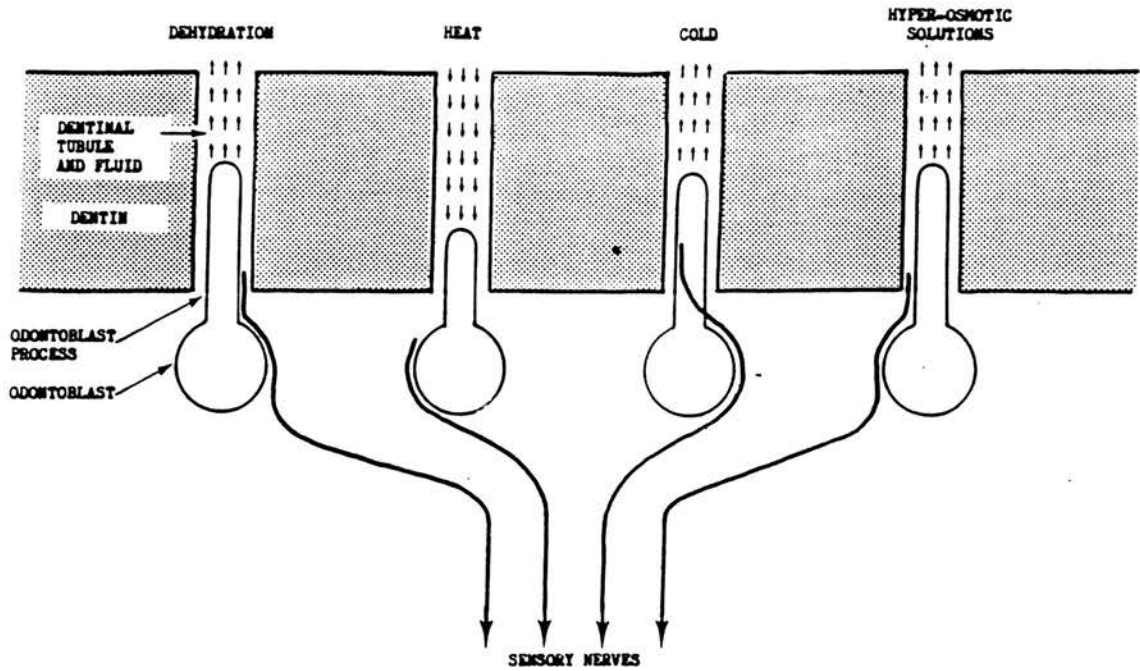
(Horiuchi & Matthews 1976, Matthews 1977). It should also be noted that the early works of Anderson and Brännström were essentially physiological studies of coronal dentine, rather than of cervical dentine sensitivity per se. Anderson & Matthews (1966) also cautioned against extrapolating their observations on non-carious healthy coronal dentine to sensitive root dentine. Another factor is the absence of a smear layer on the exposed root surface of teeth in patients complaining of sensitivity (Linden 1968, Pashley 1990).

3). The effect of thermal changes

According to Berman (1985) the perception of acute thermal stimulation can be explained by the hydrodynamic theory (**Fig. 1.2.**).

When a cold stimulus was applied to dentine, it was observed to cause a contraction of tubule contents, which in turn resulted in a rapid outward movement of fluid away from the pulp. Conversely, when heat was applied to dentine, expansion of tubule contents occurred with a subsequent increase in pressure, which resulted in a rapid inward movement of fluid towards the pulp (Brännström & Åström 1972). In a series of in vivo experiments designed to evaluate the hydrodynamic theory, Brännström & Åström (1972) observed that an elevation in temperature 30°C above ambient failed to elicit a painful response; whereas pain was invariably elicited when there was a drop in temperature. Pain elicited from prolonged application of heat is generally of a dull nature and normally took longer to develop, in contrast to the immediate sharp pain elicited from a cold stimulus (Brännström et al. 1967, Brännström & Åström 1972). Several investigators have suggested that the delay in response may be due to the larger volume of dentine which must be heated before sufficient movement of tubular contents can occur (Brännström & Johnson 1970, Brännström & Åström 1972). It is also possible that a specific pulpal temperature must be reached before pain is experienced, and this may account for delay in response (Weine 1982). According to Närhi (1990b) activation of both intradental A- and C- fibres may contribute to two different types of pain sensation

Figure 1.2.



Postulated effects of various stimuli on the movement of fluid in exposed dentinal tubules.

Illustration reproduced from Berman, L.H. (1985) Dentinal sensation and hypersensitivity. A review of mechanisms and treatment alternatives. (J. Periodont. 56, 216-222)

following heat stimulation. The pattern of nerve response appears to be a rapid A- unit action followed by a delayed C-fibre firing. The clinical features comprise an initial sharp pain following heat application and a subsequent dull pain, provided the stimulation is continued and the temperature of the pulp is elevated to about 44°C (Närhi et al. 1982a,b, Jyväskylä 1986). Rapid cooling can also induce fluid flow and cause activation of intradental A-fibres (Närhi et al. 1982a,b, Jyväskylä 1986, Jyväskylä & Kniffki 1987). Cooling may also activate A- fibres directly (Jyväskylä 1986) while C- fibres may respond to cold once the stimulus has reached the pulp (Närhi 1985a,b, Jyväskylä 1986). Activation of C- fibres, which may contribute to the dull pain induced by intense thermal stimulation of the tooth, however, appears to be associated with pulpal inflammation (Jyväskylä & Kniffki 1992, Närhi et al. 1992a,b).

More recently Kim (1986a) postulated two mechanisms whereby thermal stimuli elicit a painful response.

- 1) Based on the hydrodynamic theory, thermal stimuli evoke dentinal sensitivity by changing the physical properties of the dentine, namely, tubular radius and dentine fluid viscosity.
- 2) Thermal stimuli alter pulpal microcirculation which in turn causes sensory nerve excitation by increased tissue pressure.

These studies, therefore, support Brännström's hypothesis of a hydrodynamic mechanism (Brännström 1963a,b, 1966, Brännström & Åström 1972). Arguably the most significant fact from the Brännström & Åström (1972) study was that pain was caused by the rapid displacement of tubular contents, and not the slow outward movement of fluid which normally occurred.

According to Närhi (1985a), however, as long as the fluid flow in dentinal tubules cannot be measured in vivo, the evidence supporting the hydrodynamic theory remains unsubstantiated. Other investigators while acknowledging that the vast amount of experimental

data from both in vitro and in vivo studies appears to support the concept of a hydrodynamic mechanism of stimulus transmission across dentine; nevertheless have suggested that alternative mechanisms may also be responsible (Horiuchi & Matthews 1973, Matthews 1977, Närhi et al. 1982b, Kim 1986a,b).

Recently Vongsavan & Matthews (1991, 1992a,b), however, have demonstrated possibly for the first time (in vivo) that the velocity with which fluid flows outwards through exposed dentine can be sufficient to substantially reduce the inward diffusion of substances into the tubules. According to these investigators, it is also possible that excitation of intradental nerves by mechanical stimulation of exposed dentine may be due to a sudden interruption of an existing outward flow in the dentinal tubules. These results would, therefore, appear to substantiate the concept of a hydrodynamic mechanism as proposed by Brännström and co workers.

1.2.7. Alternative Mechanism (Modified Hydrodynamic Theory)

Desensitization by blocking nerve activity (direct ionic diffusion)

Although most studies appear to support the concept of a hydrodynamic mechanism in dentine, several investigators on the basis of conflicting experimental evidence have suggested that other mechanisms of pulpal nerve activation may also exist (Horiuchi & Matthews 1973, Matthews 1977, Närhi et al. 1982b, Kim 1986a,b).

The earlier investigations of Anderson and co-workers (1962, 1966, 1967) reported that the ability of various chemical solutions (e.g, dextrose, CaCl_2 , NH_4Cl etc) to cause pain in vivo appeared to be related to their osmotic pressure. Horiuchi & Matthews (1973), however, reported that fluid movements caused by solutions of different chemical substances could not always be (accurately) predicted by their osmotic pressure alone. Horiuchi & Matthews (1976) also applied chemical stimuli to dentinal cavities of varying depths in the cat and concluded that changes in the ionic environment around the nerve endings, rather

than osmotic pressures of the applied solutions were responsible for the induction of nerve impulses. According to Matthews (1977) the failure of 6 molal CaCl_2 to excite nerves which responded to cooling (in the dog) suggested that the fibres were not excited by an outward movement of fluid through the dentinal tubules. Both stimuli have previously been shown to cause an outward movement of tubule contents in vitro (Horiuchi & Matthews 1973). Horiuchi & Matthews (1973) also reported that application of water (at tooth temperature) to human dentine in vivo failed to cause pain, but did cause inward fluid movement in vitro. These studies would, therefore, appear to suggest that other mechanisms are responsible for the transmission of stimuli across dentine.

Several investigators have used a neurophysiological model to evaluate dentine sensitivity (Scott 1972, Edwall & Olgart 1977, Olgart 1979, Närhi & Haegerstam 1983, Närhi & Hirvonen 1987, Närhi et al. 1982a,b, 1984, 1988, Kim 1986b). The technique involves recording electrical activity from pulpal nerves following the application of hypertonic solutions. Theoretically this should produce osmotically induced fluid shifts across dentine, thereby inducing intradental nerves which may be recorded from the dentinal surface or from single nerve units dissected from the mandibular nerve.

Närhi & Haegerstam (1983) reported that application of 135 mM potassium chloride to deep cavities induced a brief burst of impulses followed by insensitivity of the nerve units to any further stimuli. More recently Kim (1986b) demonstrated in a series of studies that treatment of dentine with K^+ containing compounds (0.189 mol/L) reduced intradental nerve excitability to further chemical stimulation whereas various nitrate cation substitutes (NaNO_3 [0.25 to 2.473M], LiNO_3 [0.25 to 2.473M], 10/40% $\text{Sr}(\text{NO}_3)_2$) were ineffective. It would appear from Kim's work that the net result of raising the intratubular K^+ content is to render the intradental nerves less excitable to further stimuli by depolarizing the nerve fibre(s) membrane. Initially this increase in the K^+ content elicits an increased number of action potentials, after

the initial depolarization, however, the nerve fibres cannot repolarize due to the maintained high levels of extracellular K^+ and consequently a sustained depolarized state occurs [axonal accommodation] (Bilotto et al. 1986, 1987, 1988, Markowitz & Kim 1985, Markowitz et al. 1989, 1991).

According to Markowitz et al. (1991) the work of Närhi et al. (1982a), Panopoulos et al. (1983) and Bilotto et al. (1988) indicated that divalent cations (e.g., $CaCl_2$ [0.76M], $MgCl_2$ [0.76M], $SrCl_2$ [0.60/2.5M]) can have a dual effect on pulpal sensory nerves. For example, when high concentrations are applied to shallow cavities, divalent cations cause outward fluid movement producing transient activation of intradental nerves, whereas at lower concentrations and when applied close to the pulp via deep cavities, these solutions are only depressant.

Several investigators (Olgart et al. 1977, Olgart 1979, Gazelius & Olgart 1980) have postulated that nerve depolarization caused by elevation in intratubular potassium concentration may also lead to the release of Substance P or other neuroactive, vasoactive peptides from local intradental nerves, which subsequently modify local blood flow or nerve excitability long after the K^+ concentration has been restored to normal.

Trowbridge (1985) has also demonstrated that zinc oxide/eugenol reduced nerve excitability in the frog model. This observation has been confirmed both in vitro (Kozam 1977) and in vivo (rat phrenic nerve, Brodin & Roed 1984). Hume (1984a,b) has shown that diffusion of eugenol across dentine, 1mm in thickness was inhibitory but, at the low concentration used, not toxic to the cells; zinc oxide/eugenol when applied directly to pulp elicited an inflammatory response.

Kim's proposed mechanism of desensitization by blocking nerve activity (direct ionic diffusion) has, however, been criticised (Sena 1990).

- 1) The animal model used in these experiments needs human confirmation.
- 2) The basic assumption of the alternative mechanism is that K^+ is capable of traversing the length of the dentinal tubule in sufficient quantity and at an adequate rate to depolarize the pulpal

nerves.

There are practical difficulties in accepting this assumption. Kim's work was based on deep cut cavity preparations, with only a very thin slice of dentine between the exposed dentine surface and the pulp. In consequence K^+ had only a short distance to traverse the length of the tubule.

Secondly, in the normal clinical situation, the incoming K^+ would have to overcome the opposing pulpal pressure which produces an outward flow of dentinal fluid.

According to Mathews, as cited by Sena (1990), it is theoretically possible for sufficient K^+ concentration to diffuse across dentine and produce a concentration difference of 40mM above baseline, which would be of sufficient magnitude to depolarize the sensory nerves. Recently Orchardson & Lucas (1991), using a mathematical model to simulate the time course of K^+ diffusion along dentinal tubules, concluded that to raise extracellular potassium to levels likely to affect intradental nerve activity, a source containing 500mM would have to be applied to outer dentine for approximately 3 minutes.

Vongsavan & Matthews (1991, 1992a,b) also demonstrated (in the cat) that the outward flow of fluid through open dentinal tubules can prevent the inward diffusion of substances from the oral cavity, however, the concentration gradients were considerably less.

This modified hypothesis of dentine desensitization by blocking nerve activity (direct ionic diffusion), would, therefore appear to act, not on the basis of tubule occlusion as proposed by the hydrodynamic theory, but by reducing the sensitivity of mechanoreceptors to transient fluid shifts. It follows, that any desensitizing agent reducing nerve activity; e.g., potassium chloride/potassium nitrate, will not prevent the minute transient fluid shifts (hydrodynamic forces) from bombarding the pulp with stimuli; but they could block pulpal nerves from responding to these stimuli (Pashley 1985a).

1.2.7. Summary

Although observations from early histological studies appeared to indicate that dentine was directly innervated, it is now accepted that its coronal two thirds is largely devoid of nerve fibres apart from the predentine and its most pulpal aspects (Bernick 1948, Fearnhead 1957, Holland 1975, 1985, Lilja 1979, Ten Cate et al. 1985). Holland (1990), however, has suggested that this may not be correct in the light of recent immunohistochemical findings and improvements in fixation techniques which have demonstrated structural evidence of odontoblastic processes in peripheral dentine. The possibility that odontoblastic process may be of varying lengths and in some sites occupy the full extent of the dentinal tubule has also been proposed (Holland 1990). Takahasi (1990), however, doubted whether these new approaches to resolve this problem (e.g., the extent of the odontoblastic process in dentine) had provided any further information to that previously understood. Electrophysiological recordings in experimental animals have also indicated that intradental nerve fibres are responsible for the sensitivity of dentine, and that the endings of the responding fibres are located in the pulp border area (Närhi 1990 a,b, Närhi et al. 1992a,b). Investigators have also failed to demonstrate any morphological evidence of a synaptic relationship between odontoblast and sensory nerve endings (Byers 1979, Gunji 1982). Results from various animal and human studies have indicated that dentine sensitivity persists despite damage, disruption or destruction of the odontoblast layer, which would appear to contradict the hypothesis that the odontoblast acts as a dentinal receptor mechanism (Brännström 1962, Brännström & Åström 1964, Lundy & Stanley 1969, Ishikawa 1969, Hirvonen & Närhi 1986). If such a synaptic relationship existed, then the presence of a neurotransmitter substance such as acetylcholine would have to be demonstrated by the evidence of acetylcholinesterase activity in the dentine. No evidence has been provided to suggest that these impulses are mediated by cholinergic activity (Ten Cate & Shelton

1966).

While the exact mode of transmission of stimuli across dentine is still unclear, of the various mechanisms reviewed, the hydrodynamic theory appears to be the most commonly accepted. The earlier studies, such as those of Anderson and Brännström were essentially physiological, concerning coronal dentine rather than studies of CDS per se. Anderson & Matthews (1966) also cautioned against extrapolating their observations on non-carious, healthy coronal dentine to sensitive root dentine. Recent investigations (in the cat) by Vongsavan & Matthews (1991, 1992a,b) appears to provide evidence substantiating the hydrodynamic theory. Kim (1986a,b), however, suggested that identifying open dentinal tubules as the cause of CDS may be premature and several other investigators have also suggested, on the basis of conflicting responses to chemical stimuli, that there may be more than one mechanism involved (Horiuchi & Matthews 1973, Matthews 1977). A modification of the hydrodynamic theory, based on a neurophysiological model has been proposed by Kim (1986a,b). While the concept of dentine desensitization by blocking nerve activity (direct ionic diffusion) appears an attractive alternative to the hydrodynamic theory, this hypothesis requires further investigation (Sena 1990, Orchardson & Lucas 1991, Vongsavan & Matthews 1991, 1992a,b).

1.3. Clinical methods of assessment of Cervical Dentinal Sensitivity

Introduction

Traditionally CDS has been evaluated mainly subjectively on the basis of the individual patient's subjective response, for example, in the form of verbal rating and visual analogue scales and questionnaires. The stimuli can be grouped into four main categories: mechanical, chemical, electrical and thermal (Ong 1983).

The method and interpretation of pain assessment elicited from such stimuli, however, is open to question and interpretation. Furthermore, the subjective nature of the response and variability in patient

ability to express a given response may also complicate assessment. Currently no single method of eliciting and assessing CDS may be considered ideal.

1.3.1. Reproducibility of the stimulus

Variability in both stimuli and response to individual types of stimulus constitute major deficiencies in current efforts to monitor and evaluate CDS. In order to overcome such deficiencies the American Dental Association (1986) recommended the following study design features:

- 1) The test data should be both quantifiable and reproducible.
- 2) A critical evaluation must be made of all subjective responses. The threshold of response should be established, preferably quantified, and correlated to a clinically definable intensity. It is also recognised that the threshold is a range and not a point.
- 3) The relationship between the experimental stimulus and the defined area of hypersensitivity must be established by properly controlled clinical research.
- 4) There should be no commitment to a specific form of stimulus.
If more than one stimulus is used, then these stimuli should be reproducible and interference between them must be minimised.
- 5) Appropriate statistics should be used, and these should be justified according to the experimental design.

In addition to these study design features, the committee recommended the use of a variable stimulus level-fixed threshold response as opposed to the earlier method of fixed stimulus level-variable response for the evaluation of CDS (Kanapka 1990).

1.3.2. Subject Assessment

Pain has been described as an unpleasant sensory and emotional

experience associated with actual or potential tissue damage or described in terms of such damage (Merskey et al. 1986 cited by Melzack & Wall 1988). The diversity of the pain experience, however, explains why it has been impossible to provide a satisfactory definition of the word, pain. Melzack and Wall (1988), suggest the reason for this is that the word pain represents a category of experiences having different causes and characterised by different qualities, varying along a number of sensory, affective and evaluative dimensions. The perception of pain is based on a number of variables including the significance of pain, individual personality, psychological factors, cultural attitudes, anticipation of pain and the degree of apprehension (Mumford 1973).

Problems in evaluating the effectiveness of a desensitizing agent in a clinical trial may, therefore, derive from a lack of predictable, reliable and reproducible methodology for evaluating the subjective response of the patient, which can be further modified by social, cultural, psychological and situational factors (Ash 1986, McGrath 1986).

Verbal and non-verbal (numerical) scales as well as questionnaires such as the McGill Pain Questionnaire (MPQ) have been used to provide both qualitative and quantitative information on the subjective nature of pain following an evoked response from a painful stimulus.

According to Clark & Troullos (1990), qualitative evaluation of the subjective response in CDS clinical trials, using verbal descriptors provided by the patients themselves to describe pain, has not been documented. The patients' quantitative assessment of their own overall perception of pain associated with CDS, however, has been evaluated in clinical studies (Brough et al. 1985, Silverman 1985, Clark et al. 1987, Orchardson & Collins 1987, Minkoff & Axelrod 1987). Patients were asked to rate their own perception of overall sensitivity to hot/cold food and drink, air, toothbrushing and sweet and sour food as experienced during everyday routine. They reported using either a Verbal Rating Scale (VRS) or a Visual Analogue Scale (VAS). McGill word group

descriptors, part of the MPQ, may also be used for this purpose.

Evaluation of the subjective response following tactile, thermal, and electrical stimuli may also be recorded by the patient in the same manner.

Verbal Rating Scales (VRS)

Keele (1948) described a four point scale grading pain as slight, moderate, severe and agonising. This simple descriptive pain scale has been modified and a typical VRS may look like the following:

- 0 = No discomfort
- 1 = Mild discomfort
- 2 = Marked discomfort
- 3 = Marked discomfort that lasted more than 10 seconds

VRS offer a restrictive choice of words which may not represent the pain experience with significant precision for all patients (Huskisson 1974, Clark & Troullos 1990). The mathematical interpretation of the scoring system has also been challenged, in that the scores are often arbitrarily assigned numerical values, and the assigned scores are then analysed as if these numbers reflected true quantitative differences in pain, rather than simple qualitative differences (McGrath 1986).

Visual Analogue Scales (VAS)

A Visual Analogue Scale is a line 10cm in length, the extremes of the line representing the limits of pain a patient might experience from an external stimulus (no pain at one end and severe pain or discomfort at the other end of the line). Patients are asked to place a mark on the 10cm line which indicates the intensity of their current level of sensitivity or discomfort following application of test stimuli. VAS pain intensity can be shown either as an absolute score value or as a percentage of the maximum. The validity and reliability of the VAS for measuring both experimental and clinical pain has been demonstrated by several investigators. Clark & Troullos (1990) reported that once the

VAS procedure is properly explained to patients, it is simple to understand and suitable for use in the evaluation of stimuli response in CDS dentifrice studies. Several investigators have compared the VAS with other pain scales and the results indicate that the VAS correlates well with these methods and appears to be more sensitive in discriminating between various treatments and changes in pain intensity (Ekowski *et al.* 1972, Joyce *et al.* 1975, Ohnhaus & Adler 1975). Downie *et al.* (1978) reported that numerical rating scales (0-10) performed better than both four point descriptive scales and a continuous (Visual Analogue) scale. Scott & Huskisson (1976) demonstrated that graphic rating scales which are VAS, with descriptive terms placed at intervals along a 10cm line, may have the advantage of helping the patient decide the position of his score, especially in the absence of previous experience of pain measurement procedures, as well as enabling different subjects to record the same degree of severity of pain in the same position. These investigators concluded that this type of rating provided the best available method for measuring pain or pain relief. One objection to the graphic rating scale is that the words underneath the scale may induce a higher density of clustering of responses close to them (Seymour 1982).

Although Seymour (1982) questioned the validity of any postulated advantage to be gained by using the graphic rating scale as opposed to the plain 10cm VAS, it is apparent that the VAS can only give a uni-dimensional assessment of pain, and as such cannot distinguish between the sensory, intensity and affective (unpleasantness) aspects of pain.

McGill Pain Questionnaire (MPQ)

One of the first verbal tests which addressed the multi-dimensional nature of pain was the MPQ (Melzack 1975). The MPQ has been used to evaluate a variety of painful dental conditions including CDS. One limitation in clinical trials, however, is its complexity of vocabulary. The patient is shown 20 sets of words (**Table 1.2.**) and asked to select a word from each set which best describes present pain

experience. Each set contains up to six words in ascending order of severity. Ten of the word sets describe sensory qualities, five are affective descriptor sets, and one set describes the evaluative dimension of pain; the remaining four sets are classified as miscellaneous although they appear to be predominantly sensory. The number of words chosen provides one index (NWC), and since the words within each group set have been arranged in rank order, one can add up the total rank of all words chosen to obtain a pain rating Index (PRI). Additional information regarding the type of medication used for the pain, pain location and comparison of the present pain to previous pain experience may be obtained using the unabridged version of the MPQ. One of the advantages of the MPQ is that it provides additional data on both the qualitative and quantitative aspects of pain. Limitations of the MPQ, may, however, preclude its use in CDS studies, as it is more time consuming to administer compared to VAS and category scale procedures. The test may reflect, in part, the vocabulary limitations of the patient as well as the nature of pain per se. There may also be cultural differences in language habits which could be confounded with differences in pain expression. Patients are forced to give more consideration to the sensory aspects of pain rather than the affective or evaluative aspects in the test procedure (Chapman et al. 1985).

Several investigators (Hall et al. 1986, Zakrzewska & Feinmann 1990) have reported that the MPQ is useful in diagnosis as well as monitoring treatment outcome, although Hansson et al. (1988) reported little correlation between the MPQ and other pain rating scales (VAS, VDS and NRS) when used to evaluate CDS.

Verbal Descriptor Checklists

According to Gracely et al. (1978), Verbal Descriptor Checklists appear to allow quantitative assessment of both the sensory and affective dimensions of pain using a continuum across different pain conditions instead of words intended to distinguish conditions (syndromes).

The main disadvantage of rating scales is that pain is assumed to be

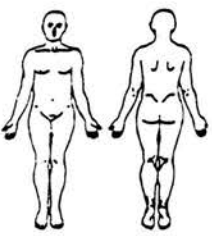
Table 1.2.

McGill Pain Questionnaire

Patient's Name _____ Date _____ Time _____ am/pm

PRI: A E M PRI(T) PPI
 (1-10) (11-16) (17-20) (21-26) (27-32)

1 FLICKERING	11 TINGING	BRIEF _____ RHYTHMIC _____ CONTINUOUS _____ MOMENTARY _____ PERIODIC _____ STEADY _____ TRANSIENT _____ INTERMITTENT _____ CONSTANT _____
2 QUIVERING	12 EXHAUSTING	
3 PULSING	13 SICKENING	
4 THROBBING	14 SUFFOCATING	
5 BEATING	15 FEARFUL	
6 POUNDING	16 FRIGHTFUL	
7 JUMPING	17 TERRIFYING	
8 FLASHING	18 PUNISHING	
9 SHOOTING	19 DRUMLING	
10 PRICKING	20 CAUSAL	
11 BURNING	21 VICIOUS	
12 DRILLING	22 KILLING	
13 STABBING	23 WRETCHED	
14 LANCINATING	24 BLINDING	
15 SHARP	25 ANNOYING	
16 CUTTING	26 TROUBLE SOME	
17 LACERATING	27 MISERABLE	
18 PINCHING	28 INTENSE	
19 PRESSING	29 UNBEARABLE	
20 GRABING	30 SPREADING	
21 CRAMPING	31 RADIATING	
22 CRUSHING	32 PENETRATING	
23 TUGGING	33 PIERCING	
24 PULLING	34 TIGHT	
25 WRENCHING	35 NUMB	
26 HOT	36 DRAWING	
27 BURNING	37 SQUEEZING	
28 SCALDING	38 TEARING	
29 BEARING	39 COOL	
30 TINGLING	40 COLD	
31 ITCHY	41 FREEZING	
32 SMARTING	42 HAGGARD	
33 STINGING	43 HAUBEARING	
34 DULL	44 AGONIZING	
35 SORE	45 DREADFUL	
36 HURTING	46 TORTURING	
37 ACHING	47 NO PAIN	
38 HEAVY	48 MILD	
39 TENDER	49 DISCOMFORTING	
40 TAUT	50 DISTRESSING	
41 RASPING	51 HORRIBLE	
42 SPLITTING	52 EXCRUCIATING	



E = EXTERNAL
I = INTERNAL

COMMENTS:

Word Descriptors McGill Pain Questionnaire (MPQ)
 Melzack, R. (1975). Reproduced from Pain 1:227-299.

a unidimensional experience varying only in intensity, and as such a broad range of psychological experience is compressed into an artificially small continuum. Patients tend to spread their responses over the entire scale regardless of the magnitude of the actual sensations (Gracely 1980). Chapman *et al.* (1985) reported a tendency for investigators to treat scores from studies as interval or ratio level scaling in statistical analysis, without evidence that patients actually use the numbers in this way. Data interpreted in this manner suggest a ranking order and imply that interval differences between the individual values are equal in magnitude, which may not necessarily be true.

Heft and Parker (1984) have shown that category scale values are not equally spaced when labelled with words commonly used to describe pain, and they advocated the use of irregular spacing, which would reflect differences in word meaning.

Price *et al.* (1983) modified VAS methodology to allow for separate assessment of both intensity and affective (unpleasantness) aspects of pain. Duncan *et al.* (1989) compared both Verbal Descriptor Checklists and the multi-dimensional VAS methodology and concluded that both VAS and Verbal Descriptors successfully quantified sensory intensity and affective aspects of pain, but that Verbal Descriptors may provide the more sensitive tool for separating intensity and unpleasantness.

The Hospital Anxiety and Depression Scale (HAD)

Recently Zakrzewska and Feinmann (1990) employed the Hospital Anxiety and Depression Scale (HAD), devised by Zigmond & Snaith (1983), in a four year clinical study in patients with atypical facial pain and trigeminal neuralgia, and concluded that the HAD scale was effective in assessing the effect of the reported pain on the wellbeing of the patient. The HAD scale does not appear to have been reported in CDS studies.

Few CDS studies have sought to assess pain intensity and unpleasantness in connection with the patient's oral hygiene activities or in

relation to suitable stimuli associated with clinical treatment (Clark et al.1985).

The patient's fear of possible discomfort from the use of a form of stimulus not normally associated with the clinical situation may also upset the reliability of subjective evaluation of the elicited response. Others, however, have concluded that reliance on subjective response alone would have minimal significance in the evaluation of CDS (Green et al.1977).

Problems still exist because of investigator inability to observe patient response to external stimuli objectively (Dayton et al.1974). Threshold measurements alone are insufficient because of variability, and because they are expressed in terms of stimulus rather than perception of pain (McGrath 1986). Variability in pain threshold from patient to patient is attributed to such factors as age, sex, cultural background, attention, suggestion, which may be further modified by various psychological variables (Woodrow et al.1972, Melzack 1973, Gracely et al.1978).

Most investigations designed to evaluate the efficacy of desensitizing agents in CDS appear to quantify response by means of criteria which may be described as objective with regard to the method per se, but in reality are subjective with regard to patient response. To some extent, the evaluation of treatment for CDS is difficult regardless of the methodology employed.

1.3.3. Mechanical (Tactile) Stimuli

Different methods of applying mechanical stimuli include scratching the dentine surface with a sharp probe (Cohen 1961, Hernandez et al. 1972, Minkov et al.1975, Zinner et al.1977, Uchida et al.1980, Carlo et al.1982, Manochehr-Pour et al. 1984, Silverman 1985, Person et al. 1989, Guo-Huo & Morimoto 1991), scaling procedures (Fitzgerald 1956, Everett 1964) as well as mechanical pressure stimulators (Smith & Ash 1964a,b, Kanouse & Ash 1969, Dayton et al.1974, Green et al.1977, Lutins et al.1984, McFall & Morgan 1985, Orchardson & Collins 1987b,

Kleinberg et al.1990) and more recently the Yeaple probe (Clark et al.1987), Minkoff & Axelrod 1987, McFall & Hamrick 1987, Silverman et al.1988, Kern et al.1989, Phantumvanit et al.1990, Prapakamol et al.1991, Sidi et al.1991).

Explorer probe use to evaluate sensitivity has been criticised. A mechanical probe introduces variability in pressure. Ideally, one would require the same tactile pressure to be exerted on all test teeth at all time intervals during a given clinical trial (Clark & Troullos 1990). The use of scaling procedures has also been criticised, being subject to such factors as pressure applied, instrument sharpness and depth of penetration. Ong & Strahan (1989) questioned whether scratching the dentine with an explorer can be considered a natural stimulus for assessment of CDS. Smith and Ash (1964a,b) developed a mechanical stimulator to provide quantitative information on patient response to scratch stimulation of dentine (Kanouse & Ash 1969, Dayton et al.1974). This device, subsequently modified (Green et al.1977) and Lutins et al.1984, McFall & Morgan 1985) incorporated a 15mm stainless steel wire with a tip ground to a fine point and capable of movement across the buccal surface of the sensitive test tooth. The scratching force could be increased by means of a small screw used to move the tip closer to or away from the root surface. The testing procedure involved moving the wire across the exposed root surface, increasing the scratching force, measured in millimetres, until a painful response (threshold value) was elicited. This device has been criticised since the stimulus intensity could not be measured in force units, and the size of the device limited its access to the labial surfaces of the anterior teeth. Smith & Ash (1964a,b) and Green et al.(1977) appear to be the only investigators who have attempted to evaluate the exact position of sensitivity on a given tooth surface, by means of an occlusal relocation key on this device.

Orchardson & Collins (1987b) developed a mechanical stimulator comprising a chuck mounted on a short metal beam which carried foil strain gauges. A sickle-shaped caries probe was mounted in the chuck at

right angles to the strain gauges. The beam carrying the strain gauges was fixed at its end to the inside of a chrome tube which formed the handle of the instrument. The probe tip was held perpendicular to the tooth which was gently scratched, with gradually increasing force, until the patient indicated that the pain threshold had been reached (minimum stimulus to evoke a sensation of pain). The device was attached to a chart recorder to register the applied stimulus in grams weight. The investigators claimed that the device afforded easy access to most tooth surfaces, with the exception of the distal aspects of second and third molars and the lingual surfaces of mandibular molars. According to Clark & Troullos (1990), this instrument appeared to provide a quantifiable and reproducible method of assessing CDS.

The Yeaple probe is an electronic pressure-sensitive device originally designed to function as a pressure-controlled periodontal probe (Polson et al. 1980). The probe was modified to accept the tine of a dental explorer (Minkoff & Axelrod 1987, McFall & Hamrick 1987, Clark et al. 1987, Kern et al. 1989). The handle of the probe is approximately the size of a fountain pen and is connected by a flexible electrical lead to a control panel. The probe is designed to deliver a pre-set force when the tip is applied perpendicular to the cervical labial surface. This force may be varied by regulating the current by means of a dial to an electromagnet controlling tip position.

Once the pre-set force is reached a red light shows on the control panel and an audible signal is activated. Application of the incremental probe settings (in gram weight) may be varied by the operator, usually in 5 gram weight steps (Minkoff & Axelrod 1987, Sidi et al. 1991), until the patient experiences discomfort. The probe setting is noted at this point. If a maximum setting of 70 gram weight is reached without any perceived discomfort, then the tooth is scored as non-sensitive. McFall & Hamrick (1987) applied settings of 25, 50 and 75 gram weight in sequence rather than in 5 gram weight increments. Teeth failing to respond at 75 gram weight were considered non-sensitive and scored 0. Clark et al. (1987) quantified pain by

determining which range setting (<20 gram weight, 20-39 gram weight, 40-59 gram weight, 60-75 gram weight) elicited a painful response. These investigators experienced problems in maintaining constant pressure on the curved surface of the cervical portion of the tooth.

The main advantage of the Yeaple probe is that tactile sensitivity can be reported in terms of a quantifiable, reproducible force (Clark & Troullos 1990). The probe tip also affords access to all tooth surfaces. One of the criticisms of the Yeaple probe is that data analysis requires an assumption that responses over 70/75 gram weight do not exist, or that no response is automatically equivalent to 70/75 gram weight. According to Ash (1986), this problem tends to defeat the use of a scaled stimulus (varied stimulus/constant response test).

Kleinberg et al. (1990) reported a hand-held scratch device, which consisted of a torsion gauge and a sharp explorer-like probe. The device was capable of easy movement across a sensitive tooth and had an indicator, displaced by the arm of the explorer tine, that recorded the force of displacement in centi-newtons. The scratch process was repeated with successively greater force until pain was perceived by the patient. The point at which pain was first perceived was considered the pain threshold. If a tooth failed to respond to a force of 80 centi-newtons it was classified as non-sensitive.

Criticisms applicable to the other methods of assessment by tactile stimuli may be relevant. The use of a sharp probe may also scratch the dentine surface. According to Pashley (1990) pressure, even from a gentle force of 5-10 gram weight, is sufficient to overcome the elastic limit of dentine, leading not only to compression and smear layer creation under the explorer tip, but also to permanent (microscopic) deformation of dentine (scratch development). This deformation of dentine may cause displacement of tubular fluid inwardly at a rapid rate, which activates mechanoreceptors, thereby triggering a pain impulse.

The scratching of the dentine may also remove a therapeutic agent deposited during a clinical trial, but this does not seem to substantially influence pain threshold (Smith & Ash 1964a,b).

One of the problems in assessing sensitivity by a scratch test is that the investigator may repeatedly miss the exact location of the sensitive site, leading to a false assumption of non-sensitivity. Several investigators have attempted to identify areas of sensitivity in both in vivo and in vitro studies (Linden 1968, Ishikawa 1969, Matsumoto et al.1980, Absi et al.1987, 1989, Yoshiyama et al.1989, 1990, Matsumoto et al.1990, Cuenin et al.1991, Oyama & Matsumoto 1991).

1.3.4. Chemical (Osmotic) Stimuli

Hypertonic solutions, for example, sodium chloride, glucose, sucrose and calcium chloride, have been used to elicit a sensation (Anderson & Matthews 1966, Miller et al.1969, Dayton et al.1974, Clark et al.1987, McFall & Hamrick 1987, Ong & Strahan 1989, Prapakamol et al.1991).

Miller et al.(1969) applied a sugared oral rinse, consisting of sweetened frozen lemon juice concentrate. Although no relevant details were published by the investigators, one may speculate whether the pH of the lemon juice influenced sensitivity by removing the smear layer.

Hypertonic solutions have been preferred to acid solutions which have a low pH and as such cause peritubular demineralisation, which could in turn aggravate sensitivity. Horiuchi & Matthews (1973) demonstrated that hypertonic solutions of sodium chloride, glucose and sucrose which elicit pain in vivo produce fluid movement through dentine in vitro. They further reported that hydrostatic pressures were more effective than osmotic pressures in producing fluid shifts. Calcium chloride has multiple effects due to its high solubility.

Superficially, it can excite intradental nerves due to osmotic movements (Panopoulos et al.(1983), whereas at deeper levels it may suppress nerve activity due to the direct effect of calcium on stabilisation of membranes (Bilotto et al.1988, Markowitz et al.1991,

Orchardson 1978, 1985).

A warm saturated sucrose solution has been utilised by several investigators (Clark et al. 1987, McFall & Hamrick 1987, Ong & Strahan 1989) as a chemical stimulus. The solution was applied with a cotton bud to the exposed dentine surface for 10 seconds, or until discomfort was perceived by the patient. Applications of hypertonic solutions to exposed dentine may exert an osmotic effect causing fluid outflow and subsequent pain. Dentinal fluid, will have a tendency to flow towards solutions of hyperosmolarity, whereas iso-osmotic solutions when applied elicit no response (Pashley 1986b). Panopoulos et al. (1983) demonstrated that while exposed dentine was not, strictly speaking a semi-permeable membrane, nevertheless the movement of tubular fluid was virtually instantaneous. Horiuchi & Matthews (1973) observed that fluid movements could not always be predicted on the basis of osmotic pressures alone. Johnson & Brännström (1974) concluded that the osmotic properties of a solution were of minor importance with regard to its pain producing effect (**see below**).

Pashley and Parsons (1987) reported that lidocaine ointment when applied to the gingivae of teeth with exposed dentine elicited pain, possibly as a result of the high polyethelene glycol concentration of the ointment. They postulated that hypertonic solutions, even if they contain local anaesthetic, elicit a pain response if the solution osmotically induces fluid movement through the dentine. The rate of diffusion of the anaesthetic molecules is slower (minutes) relative to the rate of osmotic fluid shift (seconds); hence pain is felt before anaesthesia is obtained.

Anderson and co-workers (1962, 1966, 1967, 1970) believed that hypertonic solutions were convenient quantifiable stimuli, since chemical concentration could be controlled and osmotic pressure calculated. The efficacy of chemical stimuli, however, may also be influenced by other variables, such as ionic composition, presence or absence of calcium, sodium or potassium, pH and osmolarity (tonicity) (Pashley 1986b). Närhi et al. (1988) reported that nerve responses to

hypertonic stimulation of superficial dentine were related to the osmotic pressure of the solution used. Hypertonic solutions are generally inconvenient to use and difficult to administer in a controlled manner, and may injure the adjacent soft tissues. Contamination of the tooth may also occur when hypertonic solutions are used as pain stimuli, which may, in turn, directly increase sensitivity beyond pre-test levels (Pashley 1984). Clark *et al.* (1987), however, reported no corroborative evidence to support this statement. Chemical stimuli have also been found to be unsuitable for measurement of threshold sensitivity. Anderson *et al.* (1967b) reported that repeated application of hypertonic solutions to prepared cavities in teeth reduced the sensitivity of the surface. There appear to be no studies where the pain threshold has been objectively determined by chemical stimuli.

According to Pashley (1990), Anderson and co-workers in their earlier studies were unaware of the presence and importance of the smear layer, and this, together with the low hydraulic conductance of dentine, necessitated using very large osmotic stimuli to induce sufficient fluid movement through dentine to elicit pain. Johnson and Brännström (1974) reported that a dentine surface covered with a smear layer was much less responsive to hypertonic solutions. Acid etching, for example 50% citric acid for two minutes, will reduce this layer, and consequently the hydraulic conductance of the dentine will be greatly increased (Pashley *et al.* 1981). The removal of the smear layer will, therefore, enable increased fluid flow through dentine which in turn will increase sensitivity.

This review would, therefore, suggest that recorded responses to hypertonic solutions were neither reliable, predictable nor reproducible, and as such these solutions should not be used as quantifiable stimuli in the assessment of CDS.

1.3.5. Thermal Stimuli

Sensitivity to thermal stimuli, especially to cold, appears to be the

most prevalent presenting feature in patients complaining of CDS (Harris & Curtin 1976, Kanapka & Colucci 1986, Addy et al. 1987b, Orchardson & Collins 1987a).

Cold air blast

A one second blast of cold air from a dental air syringe has been utilised in the assessment of CDS (Fitzgerald 1956, Levin et al. 1973, Tarbet et al. 1979, 1980, 1982, Uchida et al. 1980, Gangarosa 1981, Carlo et al. 1982, Manochehr-Pour et al. 1984, Silverman 1985, Clark et al. 1987, McFall & Hamrick 1987, Minkoff & Axelrod 1987, Ong & Strahan 1989, Person et al. 1989, Kern et al. 1989, Sidi et al. 1991). Cold air blasts, however, may be more useful for identifying individual sensitive teeth during screening rather than a sensitive site, since a cold air blast from a dental air syringe does not help to localise sensitive dentine (Pashley 1990). Ong and Strahan (1989) attempted to remedy this problem by using ribbon wax to isolate the sensitive dentine.

Prolonged air blasts have an unknown and possibly varying temperature effect which can be avoided by using a short application time, typically one second (Pashley 1990).

Clark and Troullos (1990) expressed concern that the range of temperature reported led to crossing back and forth over the threshold for each patient. Air blasts, however, cannot be considered graded. They are used as a constant stimulus while the investigator attempts to measure variable patient response (Pashley 1990). It is questionable whether in the absence of a stimulus of graded intensity a change in the threshold of pain can be determined.

Thrash et al. (1983) developed an electronic threshold measurement device which they claimed detected changes in sensitivity and provided a greater degree of objectivity in measuring response to a cold stimulus. This device consisted of a miniature thermistor connected to a chart recorder with an attached hand-held control for patient response. The thermistor was placed adjacent to the sensitive area for

an accurate temperature measurement of the point at which the patient first reported pain. Room temperature air (approximately 20°C) was gently blown over a sensitive site (32-34°C), until the patient registered a sensitivity threshold. Measurement of this drop in temperature was repeated three times and the average calculated. Some time, however, may be required for the test tooth to return to normal and adaptation to temperature changes may also occur (Kleinberg et al. 1990). For this reason, it is advisable that if both tactile and thermal stimuli are to be used in the same subject, the tactile stimulus should be applied before the thermal stimulus. It is also questionable whether the pain elicited in response to thermal stimuli during this procedure was due solely to cold, as the air jet would also cause dehydration (Ong & Strahan 1989).

A Yeh air thermal system was used by Minkoff & Axelrod (1987) and Silverman et al. (1988). A temperature controlled stream of air at 10 p.s.i. was directed onto the exposed dentine via a disposable plastic tip. The initial air temperature of 100°F was progressively lowered until a positive response was elicited from the patient or until the lower limit of 70°F was obtained. The air temperature was controlled by passing air from a compressor through copper coiled tubing submerged in an ice bath, to an electrical heating cartridge in the instrument's handle, where the air could be adjusted by a control device. The air temperature was continually monitored by a probe prior to exiting through the instrument tip. This technique appears to be both quantifiable and reproducible, but since the moisture content of the air jet is not controlled, it may have the disadvantage of drying and sensitizing a test tooth as the investigator proceeds down a temperature range (Clark & Troullos 1990, Kleinberg et al. 1990).

Orchardson and Collins (1987b) developed an air jet stimulator similar to the system of Thrash et al. (1983), in which the pain threshold was expressed as a reaction time. The investigators suggested that it was possible to combine surface temperature with the latency measurements to provide additional information from the same testing procedure with

the aid of a small thermistor or thermocouple, which could be incorporated into the device. The air stimulator developed a controlled jet of air ($20^{\circ}\text{C} \pm 1^{\circ}\text{C}$) from a compressed air supply. The flow of air was regulated by a flow meter which allowed air to pass into a solenoid valve, where it could either be diverted onto the tooth surface (active state) via a nozzle mounted on a transparent perspex carrier, or pass into the room air (inactive state). The device was activated when the operator pressed a foot switch which simultaneously diverted air flow to the tooth and started a three decade digital clock. When the subject experienced a barely perceptible feeling of discomfort he activated a hand held cut-off switch which automatically stopped the clock. The sensitivity was assessed by measuring the time taken for the thermal stimulus to evoke a positive response. Pain threshold was, therefore, expressed as a pain reaction time which was inversely proportional to the sensitivity.

Renton-Harper & Midda (1992) reported the use of an air jet stimulator (hypersensitivity tester machine) based on that described by Orchardson and Collins (1987b).

Recently a new microprocessor temperature-controlled air delivery system has been developed for determining cold and warm temperature thresholds of dentinal sensitivity, and used in two clinical studies (Person *et al.* 1989). This device consists of a hand held air delivery wand attached to a microprocessor-operated control unit capable of providing a temperature range of -5°C to $+85^{\circ}\text{C}$ ($\pm 0.2^{\circ}\text{C}$). Air is derived from a compressed air source and the flow regulated by a valve to maintain a constant 60 p.s.i. input to the instrument. On entering the instrument air is delivered tangentially to a vortex separator tube within the wand, where, as a result of the tube design, two distinct thermal air streams are produced. In consequence, the air emerging from the front (delivery) end of the tube is cold (-5°C) whereas the air emerging from the rear of the tube is warmer. The cold air stream enters an electrical resistance heater within the air wand and then passes into a standard dental air syringe nozzle. The temperature of

the emergent air is monitored by a thermocouple within the nozzle tip which relays this information to the micro-processor control unit. The electrical heater effects rapid and reproducible warming of the emergent airstream. A soft silicone rubber sleeve fits over the air delivery nozzle and allows placement of the nozzle against the tooth surface without discomfort to the patient, and without triggering mechanical stimuli of sensitive dentine surfaces. The air temperature settings can be adjusted in 1°C increments or decrements using the appropriate buttons on the hand held wand or in 5°C steps by successive depressions of the buttons. Conversely, the desired temperature settings can be entered on the control unit keypad. From the available data it would appear that the initial air temperature setting was 39°C, which was gradually lowered in 2 or 1°C decrements until a cold air threshold was reached when the patient perceived discomfort and raised his hand. Following an unspecified period of recovery, the threshold was reapproached for confirmation. The patients were recalled seven days later, the study teeth reevaluated and the threshold temperature values from both visits then compared. Of the 236 teeth tested, 113 (47.9%) had identical cold threshold temperatures at both visits; 31 (13.1%) had differences between + 1°C to 5°C, 15 (6.4%) of between + 6 to 10°C and 77 (32.6%) had differences greater than + 10°C. The greater cold threshold variability > + 6°C observed in 39% of teeth was attributable, according to the investigators, to inherent subject variability in sensitivity perception rather than to instrument error. For warm/hot air thresholds as recorded in study 2 (Person et al. 1989), the initial air temperature setting was at 37°C and increased in 1 or 2°C increments until a warm/hot air threshold was reached in the manner previously described for cold air threshold measurement. This technique would appear to be both quantifiable and reproducible, but the absence of any information relating to the period of recovery between each threshold evaluation and confirmation gives rise to some concern. The problem of simultaneous drying and sensitizing a test tooth as the investigator proceeds down the temperature range has been discussed

previously.

The use of prolonged evaporative stimuli has been criticised (Pashley 1990). Brännström (1960b) demonstrated that if human dentine was dried with a stream of air for five minutes, it remained insensitive to painful stimuli, as long as it was kept dry. Furthermore evaporative water loss from the dentine caused displacement of odontoblast nuclei into the tubules, although it would appear that desensitization was due to the resultant mechanical blockage (partial tubule occlusion) by the salts and organic substances left behind (Polhagen & Brännström 1971, Pashley et al. 1984a,b). In summary, the question as to whether the use of air blast stimulation can be refined to the point of providing a quantifiable method of evaluating CDS has yet to be resolved (Pashley 1990).

Cold water testing

Several investigators (Cohen 1961, Miller et al. 1969, Levin et al. 1973) have applied cold water to exposed cervical dentine. Minkov et al. (1975) applied cold water (7°C) from a syringe, while Uchida et al. (1980) utilised 20°C cold water. Flynn et al. (1985) used 15ml of cold water (7°C) which was rinsed around the mouth for a few seconds. These investigators suggested that cold water at 7°C was ideal for the identification of sensitive teeth as well as minimizing the incidence of false positive responses. Sensitivity for reasons other than CDS as discussed by these investigators, however, cannot be ruled out.

Cold water testing has also been developed to enable application of water at different temperatures to exposed cervical dentine (Johnson et al. 1982, Brough et al. 1985, Muzzin & Johnson 1989). The thermal testing technique developed by Brough et al. (1985) was modified by Muzzin & Johnson (1989) to include water at temperatures between 20°C and 0°C. The technique involved the use of disposable syringes filled with water from thermally insulated containers at 20, 15, 10, 5 and 0°C. Commencing at 20°C ($\pm 1^\circ\text{C}$), the investigators flowed water over the exposed dentine until a positive response was noted or for a maximum of 3 seconds. If

there was no response, the investigators waited two minutes and then retested the tooth with water at 15°C ($\pm 1^\circ\text{C}$). The water temperature was decreased by 5°C decrements until a positive response by the patient was obtained or until the test system limit ($0^\circ\text{C} \pm 1^\circ\text{C}$) was reached. The temperature at which a positive response was obtained or, conversely, the lack of response was recorded for each tooth tested. This method is, effectively, a threshold measurement technique.

Cold water testing, however, has been criticised for its lack of objectivity (Green *et al.* 1977). It is also difficult to determine how much water has been placed on the tooth and the timing of this placement (Gangarosa 1986). It is also difficult to control the flow of water and confine to it to a specific tooth or to a specific sensitivity locus. Furthermore, the intensity of the pain perceived by the patient at the temperature which first produced a positive response was not evaluated (Clark & Troullos 1990).

Muzzin and Johnson (1989) stated that they delayed reapplication of water for two minutes between each application of the five water temperatures in order to allow the tooth to attain body temperature. It is questionable, however, whether waiting two minutes is sufficient: up to one hour may be required before the tooth can be properly retested again by such means (Jyväsjarvi & Kniffki 1987).

Thermo-electric devices

Quantified thermal (heat and cold) stimuli have been used to determine pre- and post-treatment sensitivity levels. A thermo-electric stimulator (Naylor 1961), modified by Smith & Ash (1964a,b) has been used to report quantitative patient responses to hot and cold (Smith & Ash 1964a,b, Kanouse & Ash 1969, Dayton *et al.* 1974, Green *et al.* 1977, Addy *et al.* 1987b). It provided a continuous application of heat or cold via a probe tip small enough to allow placement on the cervical area of the tooth. The temperature of the probe tip was measured with a thermistor embedded in the tip, which enabled the current flow to cool the tip from room temperature to 12°C or conversely to heat it up to

82°C. The initial temperature for thermal sensitivity testing was set at 37.5°C. For cold stimulation the temperature was reduced in approximately 1°C decrements. At each decrement, the instrument was switched off and the stimulator tip placed in contact with the exposed root surface. This was continued until a positive response was obtained. The procedure for testing the response to heat stimuli was performed in the same manner, except that the temperature of the stimulating tip was increased from the initial temperature of 37.5°C in 1°C increments until a positive response was noted.

McFall and Morgan (1985) used a FTS Direct-Contact-Probe and measuring unit (Model DCP-80, FTS Systems Inc., Stone Ridge, N.Y.), previously used by Lutins *et al.* (1984) to measure thermal sensitivity, which was capable of providing a temperature range from -80°C to +130°C ($\pm 0.5^\circ\text{C}$). The initial temperature for testing was set at 36°C and lowered by means of an adjustable dial in 1°C decrements. At each decrement, the tip was removed from contact with the tooth for 45 seconds, and the temperature dial adjusted prior to replacing the tip on the exposed dentine. The procedure was repeated until a positive patient response was noted. The temperature at which this occurred was recorded as the threshold temperature.

Addy *et al.* (1987b), using a similar thermoelectric device to that developed by Naylor (1961), tested for response to cold stimuli by cooling the probe tip to 0°C. Teeth which gave a positive response were then restimulated with the probe set at 5°C. The procedure was then repeated at 10°C and 15°C. The sequential testing of teeth at each temperature allowed an approximately five minute time interval before the tooth was retested at the next temperature setting. It would appear that no tooth responded at the 10 or 15°C temperature settings. Ong and Strahan (1989) used a thermal probe unit developed by E.H. Davies (Institute of Dental Surgery, London) which consisted of a thermistor at the probe tip and which housed a water cooled frigistor. The thermal probe was connected by a flexible lead to circuits for temperature measurement, temperature control and constant voltage

supply units. The thermistor was capable of providing a temperature range of -5°C to $+55^{\circ}\text{C}$ ($\pm 0.2^{\circ}\text{C}$), and the device was designed to provide a suitable temperature range for eliciting a sensitivity response to thermal stimuli, via a tip (1.5mm^2 surface area of contact) suitable for placement on cervical dentine without contacting the gingival tissue. The investigators appeared to test for thermal sensitivity by utilising the extremes of the temperature range, and recorded a response following initial placement of the probe tip for up to a maximum of ten seconds. If no positive response was elicited, the probe tip was reapplied after waiting two minutes. Ong (1983) also suggested that thermal testing could be initiated at 37°C , which would give a baseline temperature threshold, and the temperature subsequently adjusted in 1°C increments or decrements until a positive response was recorded.

Thermocouple devices appear to have the advantage of precise control of temperature and to provide accurate threshold values, but unfortunately considerable time is required to set the necessary range of temperatures (Green *et al.* 1977). These devices register the temperature of the probe tip and not directly that at the tooth surface, and as such suffer from a lag between probe and tooth surface temperatures. Consequently changes in temperature must be made slowly in order that a temperature threshold of sensitivity is not bypassed (Clark & Troullos 1990). There may also be a problem with placement of a metal tip, even at body temperature, on the exposed dentine, which may trigger a painful response and consequently preclude further testing. Furthermore the heat transfer between a metal probe tip and the tooth depends on a contact area. Problems may also arise with inadequate probe contact (Person *et al.* 1989) which can result in the presentation to the tooth of poorly characterised and quantified stimuli. Criticism that these devices may not be representative of the real life clinical situation has also been made (Clark & Troullos 1990). Patients who experience CDS normally complain of cold air or cold liquids and not cold solid objects.

Most of the thermal devices presently available require contact with

the tooth surface in order to elicit a response, which means that the stimulus is both tactile (mechanical) and thermal in nature. The degree to which thermal stimuli may be considered to be tactile (mechanical) in nature has yet to be resolved (Ash 1986). Application of a water stream, however, may be considered to be almost thermal in nature as there is no pressure application. According to some investigators the use of a thermally adjusted airstream provides a no touch thermal stimulation, but unfortunately, as previously discussed, it provides both thermal and evaporative stimuli simultaneously. According to Pashley (1990), thermal stimuli should be regarded as hydrodynamic in that they induce fluid movement or pressure changes indirectly rather than by directly stimulating temperature-sensitive receptors.

1.3.6. Electrical Stimulation

According to Närhi (1985a) electrical stimulation has been used in pulp vitality testing as well as in animal experiments to identify pulp nerve units. Several investigators (Mumford & Björn 1962, Mumford 1965, 1967, 1982, Matthews & Searle 1976), however, have highlighted the technical problems associated with electrical stimulation of teeth in vitality tests. These problems include the high electrical resistance (impedance) of the hard tissues and the possibility of current flow to the surrounding structures (Närhi 1985a).

Electrical stimulation has also been used by several investigators to quantify both pre-pain and pain thresholds in CDS (Stark et al. 1977, Tarbet et al. 1979, 1980, 1982, Kleinberg et al. 1990). Unlike the other stimuli used to quantify CDS, dentinal tubule fluid movement is not necessary for transmission of the electrical stimulus, but rather the presence of lower resistance organic material in cementum, enamel or dentine (Kleinberg et al. 1990). Electrical stimuli, would, therefore, appear to be more suitable for measuring pulpal activity than for quantifying CDS (Clark & Troullos 1990).

Electrical pulp testers have been utilised to evaluate the vitality of the pulp but the validity of such pulp testing has been called into

question (Seltzer & Bender 1975a). Furthermore no correlation has been found between pain perception threshold and the histological status of the pulp (Seltzer et al. 1963). Current leakage via the periodontal ligament and subsequent stimulation of periodontal nerves may also yield false positive data. A conventional pulp tester is battery powered, producing pulses of direct current. The intensity of the output voltage (stimulus intensity) may be increased by pre-setting various numbered gradations (0-10) on a thumb wheel. Problems, however, arise in the interpretation of the information gathered in such a procedure, since it is incorrect to assume a direct relationship between stimulus intensity in volts and the number on the thumb wheel (Kanapka & Colucci 1986). Results from initial studies by these latter investigators clearly demonstrated that conventional pulp testers were not suitable for quantifying CDS.

Stark et al. (1977) developed a dental pulp stethoscope, designed to provide a range of sensitivity levels, which would aid further development of an accurate pulp testing method. The instrument consisted of a digital readout sensitive voltmeter connected to a digital printer apparatus which was activated by a push button control. A conventional battery powered electric pulp tester (Digilog) was attached to the voltmeter. The stimulus intensity was measured in volts (root-mean-square). The pulp tester tip was placed on the mid-gingival third of enamel and the tooth stimulated. A conductive gel with a pH of 5.4-5.6 was used (Ash 1986). On perceiving a tingling or warm sensation, the patient activated a hand-held point control switch which automatically stopped the stimulus and activated the recorder, which printed the voltage needed to produce a current flow that elicited the threshold stimulation. Tarbet et al. (1979, 1980, 1982) suggested there were differences in the electrical pre-pain thresholds in teeth classified as sensitive (using cold air blast) compared to non-sensitive teeth. Similar results were demonstrated by Kleinberg et al. (1990) using a modified Stark instrument, in that sensitive teeth showed both lower pre-pain and pain thresholds than healthy non-



sensitive teeth. Stark and Pelgner (1982) suggested that a value of 15 volts and above indicated a range of tooth non-sensitivity. The Stark instrument was evaluated by Tarbet *et al.* (1979) in a double-blind parallel study. The results were comparable to those obtained with the cold air blast stimulus. These investigators reported that electrical stimulation of teeth constituted an accurate and objective method for eliciting and quantifying CDS. The electrical stimulus procedure had the added advantage over the cold air blast in that the threshold stimulus could be approached slowly, so that there would be little associated discomfort. It should be pointed out, however, that despite the investigators' claims about the advantages of the pulp stethoscope, it is still a constant voltage device. The actual thresholds for nerve excitation are units of current and as such, any values given in volts may be merely a gauge of the electrical resistance (impedance) of the tooth rather than excitation of the dental nerves (Mumford & Björn 1962). In other words, the current passed at a given voltage depends upon the thickness and structure of enamel (e.g., resistance variations between a incisor and canine tooth could cause different currents for a given voltage). A threshold of 15 volts, therefore does not provide any meaningful information. Närhi (1985a) has also suggested that different fibre groups in the pulp are activated during electrical stimulation. For example, the fast conducting fibre group ($A\delta$ and $A\beta$) is the first to be activated when the intensity of the current is increased from zero. These pulp fibre units probably mediate the sensation at threshold levels or near to it (pre-pain). When the tooth is stimulated at higher levels of current, a more unpleasant and painful sensation may be perceived which according to Närhi is probably the result of the summation of action potentials of A-fibres and activation of C- fibres.

Further criticism has also been raised concerning the methodology of determining threshold values employed by Stark *et al.* (1977) and Tarbet *et al.* (1979). One of the problems of electric pulp testing is the risk of the stimulus spreading to adjacent tissues (Orchardson & Collins

1987b). To circumvent this the investigators placed the probe tip on enamel rather than on the sensitive cervical dentine and as such failed to reflect a true dentinal sensitivity. There was also the distinct possibility that by placing the probe tip on enamel the pulpal nerves were directly stimulated rather than the pulp/dentine complex, through indirect stimulation via hydrodynamic forces (Pashley 1990, Clark & Troullos 1990).

Although Tarbet *et al.* (1979) claimed that their methodology was objective, the patient was able to switch off the stimulus when discomfort was perceived. The methodology employed in this study, may, therefore, not be as objective as the investigators claimed.

The use of a constant current stimulator capable of delivering an exact current flow regardless of resistance of the hard tissues would have been a more appropriate measuring tool in these studies. According to Matthews & Searle (1976) constant voltage stimulators are unsuitable for stimulating intact teeth.

The use of electrical stimuli to quantify CDS has been criticised on the basis of being non-physiological, since the response to such stimuli fails to correspond to the painful response normally experienced by CDS patients. Pashley (1990), however, has suggested that it is theoretically possible for electrical stimuli to induce hydrodynamic fluid movement through open tubules via a phenomenon called 'electro-osmosis', which he described as the bulk movement of an electrolytic solution through a porous substance in response to the imposition of an electrical potential. Pashley concluded that in the absence of current knowledge about this phenomenon (in dentine), electrical stimulation should not be dismissed as non-physiological.

Unlike thermal stimuli, electrical stimuli are not normally encountered in real life situations, and as such there is a question as to the relationship between the voltage values obtained with the electrical stimulus procedure and the pain scale values obtained with normally experienced stimuli (Närhi *et al.* 1991).

Fear of experiencing an unknown stimulus and possible discomfort, may,

therefore, influence the patient's assessment of pain and in consequence a lower pain threshold value may be recorded. Further, stimulation of the pulp on the basis of applied voltage may fail to represent exact pain threshold values, in as much as the stimulating current depends on varying resistance pathways to the pulp or to other adjacent tissues (Ash 1986).

The use of constant current stimulators, as in neurophysiology, capable of delivering an exact current flow regardless of the resistance of the hard tissues of the tooth, has been advocated (Ash 1986, Pashley 1990).

Furthermore, because current flow is the critical variable in stimulating nerves, Pashley (1990) considered the use of constant current stimulators essential in the study of nerve thresholds and sensitivity, although ideal stimulators of this type do not appear to have been used for the assessment of CDS in clinical studies.

1.3.7. Application of test stimuli

The mode and sequence of applying a stimulus which can be varied in intensity is important. Ash (1986) suggested that an increase or decrease in the level of heat or increase in the level of electrical energy should be monotonic rather than delivered in a random order approach. He concluded that while a continuous increase may not be possible, both incremental as well as continuous increases or decreases in stimulus intensity should occur within a standard time frame.

The order of application when more than one kind of stimulus is used is important. Care should be taken to ensure that the first should not distract from the second, nor the second from the third and so on. The least disturbing stimulus should, therefore, be applied first, with the most disturbing used last (Ash 1986, Clark & Troullos 1990). Several investigators have applied either tactile, electrical or heat stimuli prior to the application of cold air on the basis that the former do not appear to elicit a painful response which could affect the latter (Tarbet et al. 1979, 1980, 1982, Minkoff & Axelrod 1987, Orchardson &

Collins 1987b, Addy et al.1987b, Kern et al.1989, Person et al.1989). The applied stimulus must be reproducible and behaviour predictable. Without such quantification it is difficult if not impossible to compare the findings of different investigators (Ash 1986). No method of evaluation, however, may be considered reliable when used alone (Addy & Dowell 1983, Ong & Strahan 1989). There is plainly a need to investigate the measurability and reproducibility of these stimuli using methodologies and instrumentation more related to the clinical situation.

1.3.8. Summary

This review of the literature indicates that there are problems in evaluating patient subjective response to the various stimuli used in the assessment and treatment of CDS. Opinions vary as to the reliability of some of these methods of assessment (Green et al.1977, Addy & Dowell 1983, Lecointre et al.1986, Addy et al.1987b), although more recently efforts have been made to develop controlled reproducible stimuli more suited to the evaluation of CDS (Silverman 1985, Minkoff & Axelrod 1987, McFall & Hamrick 1987, Addy et al.1987b, Clark et al. 1987, Ong & Strahan 1989, Kern et al.1989, Person et al.1989, Sidi et al. 1991).

Currently no single method of eliciting and assessing CDS may be considered ideal. The plethora of devices covered in this review would also suggest that no one device is universally accepted as the ideal method for assessing CDS. The absence of suitably objective methodology of assessing CDS and the lack of standardised measurement of the subjective response following application of stimuli, therefore, still gives cause for concern.

Further research, is therefore, required to evaluate suitable methodology for the quantification of realistic test stimuli under controlled clinical conditions, whereby the subjective response may be satisfactorily measured.

1.4. Management of Cervical Dentinal Sensitivity

Introduction

According to Dowell et al. (1985) and Addy et al. (1985) the management of patients suffering from CDS should be based on the following:-

1) Correct diagnosis of the condition by:

- a) The identification of zones of exposed dentine which when suitably stimulated produce pain.
- b) The identification of those factors which expose the dentine and could be related to the opening of the contained tubules.
- c) The elimination of other causes of pain either as separate entities or co-existing with CDS.

2) Prevention or removal of aetiological factors by:

- a) Advice on correct non-traumatic toothbrushing technique.
- b) Dietary counselling with respect to the intake (especially frequency) of acidic fruits and beverages, particularly as they relate to times of toothbrushing.
- c) Elimination of other habits or disease which cause dentine exposure.
- d) The use of fluoride mouthrinses which may reduce the effects of aetiological factors by protecting against acid solubility.

3) Therapy considered on the basis of severity of the problem

- 1) For isolated problems, therapy is largely professionally delivered and should be directed towards varnishes, adhesives, filling materials and cervical restorations.
- 2) For general sensitivity, suitably formulated dentifrices preferably having an affinity for dentine such as silica and containing fluoride or strontium may be used.
- 3) In severe sensitivity root canal therapy or the extraction of the offending tooth (Ong & Strahan 1989) might be indicated.

1.4.1. Desensitizing Agents and Techniques

Numerous desensitizing agents have been utilised in an attempt to alleviate CDS. These therapeutic agents have been broadly classified into the following groups:-

- 1) Anti-inflammatory drugs
- 2) Protein precipitants
- 3) Tubule occluding agents
- 4) Tubule sealants
- 5) Miscellaneous

(Modified from Ong 1986)

Recently Scherman & Jacobsen (1992) have suggested that treatment modalities fall into two main categories, chemical and physical, based on their supposed mode of action. Neither the Ong (1986) nor the Scherman & Jacobsen (1992) classifications are ideal, they are somewhat imprecise and fails to take into account the action of agents which are said to block pulpal nerve activity by direct ionic diffusion. For the purposes of this literature review, however, a modified Ong (1986) classification is used.

According to Grossman (1935) an ideal desensitizing agent or technique should fulfil the following criteria:-

- 1) It should not unduly irritate, nor in any way endanger the integrity of the pulp
- 2) It should be relatively painless on application or shortly afterwards
- 3) It should be easily applied
- 4) It should be rapid in its action
- 5) It should be permanently effective
- 6) It should not discolour tooth substance

1.4.2. Anti-inflammatory drugs

Several investigators have utilised topically applied corticosteroids

based on the rationale of suppressing pulpal inflammation to reduce sensitivity from both exposed coronal and cervical dentine (Fry et al. 1960, 1965, Bowers & Elliot 1964, Lawson & Huff 1966, Stanley et al. 1965, Swerdlow et al. 1965, Gurney 1970). Langleland et al. (1971), however, found that corticosteroids did not prevent or reduce pulpal inflammation, resorption or apposition of dentine. Topical application of 1% prednisolone has been reported to reduce thermal sensitivity (Mosteller 1962, Bowers & Elliot 1964, Dachi & Stigers 1967, Gurney 1970, Rosen & Stallard 1980). The Mosteller study was uncontrolled, with teeth prepared for gold restorations, although a subsequent controlled study (Mosteller 1963) in which half the teeth were treated with prednisolone and half used as controls, reported that, whereas prednisolone reduced thermal sensitivity (46°F water onto teeth; 50% of untreated teeth remained sensitive), it did not eliminate pulpal inflammation. Histologically, treated teeth revealed only slight to moderate pulpal changes, whereas controls disclosed marked to severe pulpal injury. Mosteller considered that one of the constituents of the prednisolone solution, the vehicle, may have had a slight desensitizing effect. A subsequent study (Stanley et al. 1965) tested the steroid component against the vehicle, camphorated parachlorophenol and metacresyl acetate. While prednisolone alone was more effective than the vehicle initially, the inflammatory response after twelve days was similar in both groups. The investigators suggested that prednisolone and the vehicle acted synergistically, with prednisolone modifying the initial response and the vehicle maintaining the altered response. Rosen and Stallard (1980) evaluated Prednisol Plus, a combination of 1% prednisolone and the vehicle, parachlorophenol and metacresyl acetate, against placebo and individual constituent solutions. The results were difficult to assess, since both intracoronal restorations and full crown preparations were utilised in each group. The investigators concluded that Prednisol Plus decreased post-operative sensitivity to percussion, electrical stimulation, cold and heat more than the others and suggested that it would be a valuable adjunct as a lining in

restorative procedures. Although no figures were provided, the application of a Copalite varnish or base, following steroid therapy to simulate the normal clinical situation, may have influenced the results. Dachi and Stigers (1967) reported that copal varnish following steroid application (to cavities) was more effective in reducing thermal sensitivity than prednisolone alone.

As regards mechanism of action Mjör (1967) demonstrated that steroids induced localised peritubular mineralisation which, according to the hydrodynamic theory, would result in reduced tubule fluid flow and a subsequent decrease in dentinal sensitivity. Mjör and Furseth (1968) also reported irregularly shaped tubules close to the predentine, many obturated by a highly electron dense material. As the steroid was left in contact with the dentine from forty one to ninety five days, the relevance of this study to the clinical management of CDS is questionable (Trowbridge & Silver 1990).

The exact mechanism of steroid-induced mineralisation, however, has yet to be ascertained (Krauser 1986). The validity of using corticosteroids to treat CDS has been questioned (Ong 1986), although Seltzer and Bender (1990) suggested there would be no damage to the pulp from a single application to dentine.

The role of pulpal inflammation in the aetiology of CDS remains unclear, although it is possible that plaque products overlying exposed dentine may diffuse through open tubules to the pulp where they may elicit an inflammatory response (Bergenholtz 1977, Bergenholtz *et al.* 1982, Bergenholtz & Lindhe 1978, Warfringe *et al.* 1985). Recently several investigators (Kim 1990, Kim *et al.* 1992, Liu *et al.* 1992, Olgart 1990) have suggested that repeated stimulation of sensitive teeth (in the animal model) may induce pulpal changes. Such changes could occur through induction of neurogenic inflammation and its subsequent effect(s) on pulpal blood flow. Presumably if there is a decrease in pulpal blood flow, there would be a subsequent reduction in the outward fluid flow which would be insufficient to flush out any metabolites from the pulp, as well as preventing the inward diffusion of plaque

metabolites through the open dentinal tubules from the oral environment. The combination of bacterial products and endogenous mediators of inflammation may subsequently lower the pain threshold to the point where previous sub threshold stimuli become sufficiently strong to cause pain (Trowbridge 1986, Kim 1990, Kim et al. 1992, Markowitz & Kim 1992). The resultant inflammation may either contribute to spontaneous desensitization or on rare occasions create irreversible pulpal damage. Evidence to support such a hypothesis appears to be lacking (Trowbridge & Silver 1990). Collaert and Fischer (1991), however, have cautioned against extrapolating the results of studies in coronal dentine (e.g., experimental cavity preparation and direct diffusion of plaque metabolites through to the pulp) to cervical dentine and the symptoms associated with CDS.

There is a need for further investigation of steroids in the management of CDS, since most relate to their application following cavity preparation, rather than to cervical dentine. These studies mainly claim that relief was immediate, but provided little evidence to suggest that this desensitization was due to the action of the steroid alone.

1.4.3. Protein precipitants

As the odontoblast and its process was believed by several investigators to participate in the transmission of sensory stimuli, so-called protein precipitants, e.g., silver nitrate, zinc chloride and formaldehyde, were used to block transmission by denaturation of cell and process. Recent studies, however, suggest that the odontoblast does not possess the properties of a sensory receptor (Kroeger et al. 1961, Matthews 1970), and that the dentine remains sensitive following experimental destruction of the odontoblast layer (Brännström & Åstrom 1964, Brännström 1966, Lundy & Stanley 1969, Lilja et al. 1982). Greenhill and Pashley (1981) observed in vitro that protein precipitants decreased hydraulic conductance of dentine (Lp) in sections in which the tubules were devoid of odontoblastic processes,

which would suggest that the protein in the tubules was not a determining factor in the reduction of dentine permeability.

1.4.3.1. Zinc chloride-potassium ferrocyanide

Gottlieb (1935) developed a zinc chloride-potassium ferrocyanide impregnation method for desensitizing root surfaces and cavities, with varying effectiveness (Gottlieb 1947, Everett 1964, Everett et al. 1966, Reynolds 1968). The procedure involved a 40% solution of aqueous zinc chloride which was rubbed into the exposed dentine surface and allowed to remain for one minute. This was followed by a 20% aqueous solution of potassium ferrocyanide which was burnished onto the dentine surface until an orange precipitate, presumably zinc ferrocyanide, was formed. Subsequent SEM investigation revealed a dense highly crystalline deposit over the dentine surface, although, as the crystals were rather large, it would appear unlikely that they penetrated the tubules (Greenhill & Pashley 1981). This would, however, indicate a tubule occluding rather than a protein precipitating action. Trowbridge & Silver (1990) expressed doubt as to whether this method could provide a more efficient means of desensitizing dentine than burnishing alone. Reynolds (1968) observed that zinc chloride solution was caustic and could irritate both soft tissues and bone.

1.4.3.2. Silver Nitrate

Silver nitrate has been used alone and in combination with other agents, e.g., formaldehyde, in the treatment of CDS, because investigators assumed that it had the ability to cause protein precipitation within the tubules and thereby decrease sensitivity (Seltzer & Bender 1975b). However, its use was limited to posterior teeth as it caused a black discolouration (Grossman 1935, Everett 1964, Everett et al. 1966, Reynolds 1968). Reynolds (1968) reported that the silver reacted with the proteins in the tubules causing precipitation and subsequent obliteration. Greenhill and Pashley (1981) observed that silver alone or in combination with formalin precipitated silver

chloride or elemental silver respectively, which greatly reduced fluid flow in the dentine disc model. Several investigators, however, have suggested that silver salts can diffuse through the dentine into the pulp resulting in slight pulpal inflammation (Wycoff 1982, Kleinberg 1986). Anderson and Matthews (1966) and Naylor (1968) measured dentine sensitivity before and after silver nitrate application and found no significant difference in response to a thermal stimulus.

According to Anderson & Matthews (1966) protein precipitants such as silver nitrate do not decrease desensitization of dentine to osmotic (chemical) stimuli.

1.4.3.3. Formaldehyde (Formalin)

Formaldehyde as formalin in water has been used at full strength (40%) topically and in various dilutions in both mouthwashes and dentifrices in the treatment of CDS (McFall 1986). Several investigators have claimed that may act either as a protein precipitant or a tubule occluding, although there is no evidence to support this statement.

One of the earliest proponents of topical application of 40% formalin in the treatment of CDS was Orban (Everett *et al.* 1966), although formalin at this strength was considered injurious to the oral mucosa. The use of a 20% formalin mouthwash was also advocated, although caution was advised with regard to possible mucosal hypersensitivity reactions (Everett *et al.* 1966). Other investigators have noted hypersensitivity reactions (Addy & Dowell 1983, Yankell 1982, Grossman 1935, Fitzgerald 1956, McFall & Morgan 1985). According to Everett (1964) and Everett *et al.* (1966), Gottlieb and Orban (1933) also advocated a 2% paraformaldehyde dentifrice. Grossman (1935) proposed formalin as the medication of choice for the treatment of anterior teeth as, unlike silver nitrate, it did not stain the teeth. Fitzgerald (1956) reported that a 1.4% formalin dentifrice relieved discomfort, a finding confirmed by others (Abel 1958, Toto *et al.* 1958, Burman & Goldstein 1961, Burman 1963, Forrest 1963, Kimmelman *et al.* 1969). Most of these findings, however, were based primarily on subjective

response. Subsequent studies reported generally negative results. Smith and Ash (1964a) used standardised tactile and thermal stimuli in a double-blind study and reported no significant difference between placebo and formaldehyde after sixty days of dentifrice use. Others reported that the dentifrice did not differ from a control in relieving CDS (Bolden et al.1968, Hazen et al.1968, Tarbet et al.1982). More recently McFall and Morgan (1985) in a four week double-blind study involving 67 patients used a combination dentifrice containing 1.3% formalin and 0.8% sodium monofluorophosphate. They demonstrated a statistically significant reduction in sensitivity to a controlled thermal stimulus at 28 days when compared with a control dentifrice. No significant difference was observed in response to a controlled mechanical stimulus. The investigators, however, recognised the limitations of a study comparing a dentifrice containing two possible active ingredients with a control dentifrice containing neither. The abrasive filler (calcium carbonate) may have been responsible for any occlusion of the tubules (Greenhill & Pashley 1981). Addy et al. (1987a) compared a formaldehyde dentifrice (Emoform) with strontium chloride hexahydrate (Sensodyne) and three non-commercially available silica-based dentifrices. They concluded that following six weeks of dentifrice use and a further six week evaluation, the formaldehyde dentifrice showed no significant improvement by semi-quantitative (cold air) and quantitative (thermo-electric device) methods of assessment, although there was some subjective benefit. These in vivo findings were consistent with previous in vitro studies which demonstrated that a formaldehyde containing dentifrice had little or no effect on the dentine surface (Addy & Morgan 1982, Mostafa et al.1983). Greenhill and Pashley (1981) also reported that 10% formalin was relatively ineffective in reducing the hydraulic conductance of dentine in vitro.

Although formaldehyde has been claimed to achieve its clinical effects as a protein precipitant by precipitating salivary protein in the dentinal tubule, this is considered unlikely according to Addy and Mostafa (1988) who failed to observe any changes after four weeks

repeated saliva/formaldehyde treatments in vitro. The exact mode of action remains unclear. Results from both in vitro and in vivo studies indicate that formaldehyde has little or no effect in relieving CDS.

A formaldehyde containing dentifrice (Thermodent) which had been evaluated in the Tarbet et al. studies has since been replaced by SCH (Devaney 1982).

1.4.3.4. Strontium chloride hexahydrate (SCH)

SCH has been claimed to act both as a protein precipitant and a tubule occluding agent (Cohen 1961, Skurnik 1963, Blitzler 1967, Gedalia et al. 1978, Uchida et al. 1980). Gutentag (1965) also demonstrated that strontium may stabilise excitable neural membranes by modifying their permeability to sodium and potassium. Although the exact mode of action of the strontium ion is unclear, several investigators have shown that SCH causes deposition of an insoluble barrier, possibly a calcium strontium-hydroxyapatite complex, at dentinal tubule orifices (Pawlowska 1956, Ross 1961, Blitzler 1967, Gedalia et al. 1978), whilst Kun (1976) demonstrated in vitro that SCH ions produced significant penetration of dentine.

Further in vitro studies (Greenhill & Pashley 1981, Mostafa et al. 1983, Pashley et al. 1984c, Addy et al. 1990a), however, suggested that these results were attributable, not to the active ingredient, but to the abrasive component which may contribute to the formation of a smear layer and to some degree occlude the exposed dentinal tubule orifices, as observed in the dentine disc model system (Pashley 1984, 1986a,b, Pashley et al. 1987).

Pashley and Galloway (1985b) reported the effects of a two minute application of 5% potassium nitrate (KNO_3), 10% SCH, 2% sodium fluoride (NaF), 20% silver nitrate (AgNO_3) and potassium oxalate ($\text{K}_2\text{C}_2\text{O}_4$) solutions on dog dentine permeability in vivo and concluded that neither 5% KNO_3 nor 10% SCH produced any significant reduction in dentine permeability. These investigators did not rule out the possibility that these agents may desensitize dentine via neural

effects unrelated to hydrodynamic mechanisms.

SCH has been widely used in dentifrice form (10% SCH) for the treatment of CDS (Meffert & Hoskins 1964, Blitzler 1967, Shapiro *et al.* 1970a,b, Carrasco-P 1971, Hernandez *et al.* 1972, Uchida *et al.* 1980, Collins *et al.* 1984, Minkoff & Axelrod 1987). Although a number of these studies have demonstrated improvement ranging from 30-80% reduction in sensitivity when compared to other dentifrices and placebo, the results are conflicting and somewhat difficult to interpret, due in part to different methodologies and patient criteria. Smith and Ash (1964b) using standardised thermal and mechanical stimuli reported no significant improvement with either SCH or placebo dentifrice at thirty or sixty days compared to baseline. Shapiro *et al.* (1970a,b) reported that SCH was only equal in efficacy to sodium monofluorophosphate (MFP); whereas Hernandez *et al.* (1972) reported that SCH was less effective than MFP dentifrice, although significantly better than placebo.

Singh *et al.* (1984) compared SCH with MFP, formalin and placebo, and reported that SCH alleviated discomfort to mechanical, hot and cold stimuli to a greater degree. Zinner *et al.* (1977) reported that SCH was less effective than sodium citrate pluronic gel, Wei *et al.* (1980) failed to demonstrate a statistical difference between SCH and placebo in a six week double-blind clinical study. Tarbet *et al.* (1982) reported that SCH was less effective than KNO₃ in relieving sensitivity in a four week clinical study, although Collins *et al.* (1984) in a twelve week study demonstrated that both were effective in reducing tactile sensitivity. Both SCH and KNO₃ were effective in reducing sensitivity to thermal (cold) stimuli, although KNO₃ appeared more effective in reducing the number of sensitive teeth. Silverman (1986) also compared SCH and KNO₃ over a 22 week period and reported favourable responses to a variable force probe and variable air temperature for both dentifrices. Other investigators (Clark *et al.* 1985, Addy *et al.* 1987b), however, have questioned the effectiveness of SCH in reducing CDS. Recent studies (Addy *et al.* 1987a, Jackson *et al.* 1989, 1990) comparing

a silica-based product containing strontium acetate and fluoride (SrAc_2F) with Sensodyne containing SCH and the abrasive diatomaceous earth reported that the SrAc_2F dentifrice was more effective. The efficacy of SCH dentifrices in the treatment of CDS, however, was demonstrated in a twelve week double-blind comparative (placebo) clinical trial in which the levels of sensitivity were assessed by three methods of assessment, namely, thermally controlled cold air stimulus (Yeh device), tactile stimulus with an electronic pressure-sensitive probe (Yeaple probe) and subjective response (Minkoff & Axelrod 1987).

1.4.4. Tubule occluding agents

Currently the most widely accepted hypothesis for CDS is the hydrodynamic theory of dentine sensitivity (Brännström & Åström 1972). It follows that any agent which can reduce the minute hydrodynamic fluid shifts within tubules should in theory reduce CDS. The concept of tubule occlusion as a method of dentine desensitization is a logical extension of this theory (Pashley 1986a). Many of the desensitizing agents used to treat CDS have also been shown to be effective in reducing dentine permeability in in vitro studies (Greenhill & Pashley 1981, Pashley et al. 1984c, Takahashi 1986), which would appear to support the hydrodynamic theory as a basis for CDS. Not all desensitizing agents, however, reduce CDS by tubule occlusion, e.g., KNO_3 , and it would appear that an alternative mechanism of action in which pulpal nerve action is blocked by alteration of Sensory Nerve Activity [SNA] (direct ionic diffusion) may also be involved (Kim 1986a,b).

1.4.4.1. The effects of burnishing dentine

Several desensitizing agents have been applied to exposed dentine by burnishing with an orange stick, metal plastic instrument, rubber cup or dental tape (Lukomsky 1941, Hoyt & Bibby 1943, Hiatt & Johansen 1972, Murthy et al. 1973, Tarbet et al. 1979, Overman 1983). A paste

containing 33% NaF, kaolin and glycerin has been claimed to be effective in reducing CDS by this means (Lukomsky 1941, Hoyt & Bibby 1943, Murthy et al. 1973, Tarbet et al. 1979). None of these investigators, however, attempted to evaluate separately factors of the effects of paste and burnishing. All lacked a burnishing control, or one in which the exposed dentine was burnished with a fluoride-free paste and, apart from the Tarbet study (1979), placebo pastes and blinded methods were not employed. Pashley et al. (1987) in an in vitro study compared burnishing alone, kaolin in glycerin, NaCl in glycerin, NaF in glycerin and the complete NaF/kaolin/glycerin paste. NaF was no more effective than NaCl or kaolin, suggesting that the important variable was the act of burnishing and not the presence or absence of a specific ingredient. Pashley (1984) also demonstrated that burnishing dentine dry with an orange stick reduced dentine permeability by 70%, by producing a smear layer on the surface which partially occluded the tubule orifices. Hiatt and Johansen (1972) reported that burnishing produced a highly polished root surface which was clinically insensitive.

The use of pure glycerin has been advocated for the treatment of CDS (Colaneri 1952) and, according to Hastreiter (1989), 0.4% stannous fluoride gels (SnF_2) contain 98% anhydrous glycerin, which may have contributed to the claimed reduction in CDS in clinical trials involving SnF_2 . Miller et al. (1969), however, showed that SnF_2 gel was better than a glycerin placebo gel; although one third of the patients experienced some relief with the plain glycerin gel. A placebo effect cannot be ruled out. More recently Reinhart et al. (1990), in a four week pilot study involving 12 patients, reported that glycerin gel produced a sustained decrease in sensitivity to a (cold) thermal stimulus.

The precise mode of action of glycerin is unknown, although Reinhart et al. (1990) postulated an effect on pulpal nerves at the dentine pulp border by alteration of the calcium or potassium concentration in dentinal and pulpal fluid, which in turn would decrease neural

activity; although no evidence for this mode of action was presented by these investigators. These investigators also postulated that as glycerin is a hydrophilic agent it could simply dessicate the dentinal tubules, reducing their diameter, thereby reducing dentinal permeability and fluid flow. No evidence was provided to support this claim. Both burnishing (Pashley et al. 1987) and glycerin (Colaneri 1952, Reinhart et al. 1990) have been reported to elicit discomfort on application.

1.4.4.2. Aluminium lactate

Several investigators have utilised aluminium lactate (Mayer 1964, Uchida et al. 1975, Takahashi 1986, Phantumvanit et al. 1990, Horiuchi 1991, Prapakamol et al. 1991). Mayer (1964) reported that a dentifrice containing 1% aluminium lactate and 1% aluminium fluoride (pH 3.5) had a desensitizing effect. Uchida et al. (1975), however, failed to confirm this using the same dentifrice, evaluated by a response to a tactile stimulus, although they did report relief from pain induced by thermal stimuli (air and water). An in vitro study (Takahashi 1986) reported that a 2.18% aluminium lactate-containing dentifrice (pH 7) appeared effective. Takahashi demonstrated that 1% aluminium lactate (pH 3.5) as in the Mayer and Uchida studies produced a minimal effect on permeability, and postulated that the effect was due to a precipitated product of aluminium fluoride and dentine at the acidic pH 3.5 value. He asserted that pH 7 was optimal for reducing permeability with aluminium lactate. A further clinical study cited by Takahashi (1986) claimed that a 2.18% aluminium lactate dentifrice was highly effective, although no details were provided. More recently, Phantumvanit et al. (1990) compared a 2.18% aluminium lactate dentifrice with a SCH dentifrice and placebo in an eight week double-blind comparative parallel study involving 60 patients, and reported that in response to a tactile stimulus (electronic pressure sensitive probe) the aluminium lactate group was only more sensitive at an early stage of treatment, Prapakamol et al. (1991) in an eight week double-blind clinical study

involving 120 patients compared aluminium lactate, SCH and placebo dentifrices using mechanical, thermal and chemical stimuli (Yeaple probe, cold water 0-25°C, and 4M sucrose solution) and concluded that aluminium lactate significantly reduced CDS compared to SCH and placebo groups when tested with mechanical and chemical stimuli. No significant differences were observed between groups for thermal stimuli.

The available evidence would indicate that aluminium lactate has tubule occluding properties, although according to Sena (1990), this evidence has not been fully substantiated in either in vitro or in vivo studies.

Recent epidemiological studies have suggested a correlation between the frequency of Alzheimer's disease and the aluminium content of drinking water, and concern has been expressed with regard to aluminium containing dentifrices as they may contribute substantially to the ingestion of aluminium from other sources (Driessens et al.1991).

1.4.4.3. Calcium-sucrose phosphate-calcium orthophosphate complex (CSP)

CSP, a complex mixture of calcium salts of phosphoric acid, esters of sucrose and inorganic calcium orthophosphate (Craig 1973), was previously used as a cariostatic agent (Harris et al.1967, 1968, 1969, Rogerson 1973) and in a dentifrice (10%) or as a 40% gel to reduce CDS (Craig 1973, Harris & Curtain 1976). These studies, however, lacked proper controls. Results were based on a study involving 12 patients and a collection of clinical observations from 32 participating dentists. Both studies relied on patients' subjective response. The exact mode of action of CSP is unclear, although in vitro studies on dissolution of hydroxyapatite and hardening of human tooth enamel by sugar phosphates demonstrated that CSP could not only reduce dissolution but also effectively remineralize enamel. This rehardening process was rapid when CSP was incorporated in a dentifrice (Napper & Smythe 1966, Brady et al.1966, 1968, Lilienthal et al.1968).

1.4.4.4. Strontium Chloride Hexahydrate (SCH)/Formaldehyde

(See section 1.4.3.)

1.4.4.5. Calcium Hydrophosphate (Ca[HPO₄])

Electron microscopic and microradiographic studies demonstrated that Ca[HPO₄] burnished into exposed dentine obturated dentinal tubules. These deposits appeared to consist of small particles of mineralized dentinal matrix and Ca[HPO₄] crystals (Hiatt & Johansen 1972).

Permeability studies with trypan blue also demonstrated that when Ca[HPO₄] was burnished into dentine, the dye was blocked, whereas untreated surfaces as well as untreated teeth with class V cavity preparations were readily stained. A clinical trial by these investigators involving an experimental group of 108 patients (Ca[HPO₄]/burnishing) and a control group of 20 patients (burnishing only) reported that 93% of patients in the treated group claimed relief of pain, compared to only 25% of patients in the control group. There are difficulties, however, in accepting the conclusions of this study for several reasons. The investigators relied mainly on the patient's subjective response to pain following treatment. No baseline or post-treatment values were provided and no statistical analysis of the data was published following the completion of this non-blind study, which appeared to have lasted for only about one week.

Overman (1983) compared Ca[HPO₄] with a distilled water placebo in a four week double-blind study using a split mouth design. The study involved eight post-treatment periodontal treatment patients, and reported that Ca[HPO₄] significantly decreased response to tactile and thermal stimuli. Evaluation of this study is difficult for several reasons. The number of patients involved was very small and the selection of those who had recently completed periodontal treatment may be questioned. It is conceivable that the reported reduction in CDS was related to the occurrence of a natural desensitization over time (Karlson & Penney 1975), rather than to any efficacy on the part of the desensitizing agent. An associated placebo effect has been reported in

clinical trials, which may also account for the observed reduction in sensitivity. It is worth noting that the distilled water group showed an improvement in the Overman study. Levin et al. (1973) also reported a 40% relief in sensitivity after six months treatment with distilled water.

In the Overman study (1983), one drop of 7°C water was placed on the exposed dentine surface and an explorer used to test for response to thermal and tactile stimuli.

Problems with regard to the differing methodologies have been discussed elsewhere (**section 1.3.**).

Hiatt and Johansen (1972) claimed that the beneficial effect of $\text{Ca}[\text{HPO}_4]$ was lost if good oral hygiene was not maintained, and suggested that in the presence of accumulated plaque there was preferential dissolution of $\text{Ca}[\text{HPO}_4]$ crystals.

Further research into the long term efficacy of $\text{Ca}[\text{HPO}_4]$ as a desensitizing agent has been advocated (Overman 1983), but to date no published data have been forthcoming.

1.4.4.6. Sodium citrate and pluronic gel (127)

A dentifrice containing 2% dibasic sodium citrate pluronic F 127 gel has been reported to have a beneficial effect in the reduction of CDS (Zinner et al. 1977, Wei et al. 1980, Collins & Perkins 1984, McFall & Hamrick 1987). Evidence from other clinical studies, however, has been somewhat inconclusive (Tarbet et al. 1982, Clark et al. 1987, Ong & Strahan 1989). Zinner et al. (1977) in a six week double-blind study involving 168 patients used a subjective tactile stimulus to compare sodium citrate pluronic F 127 gel with a 10% SCH dentifrice, 0.4% SnF_2 in anhydrous glycerol, a pluronic F 127 gel and a placebo control dentifrice and reported that the number of sensitive tooth surfaces was reduced in all groups including the control. The sodium citrate pluronic F 127 gel demonstrated 84% improvement compared with 60% improvement in the control group. The pluronic F 127 group (without sodium citrate), however, also showed significant improvement (74%)

suggesting that it was the pluronic F 127 gel formulation that was important rather than the sodium citrate (Kanapka 1990). Patients using SnF_2 or SCH dentifrices did not appear to exhibit significant improvement over the control group. Wei *et al.* (1980) in a six week double-blind study involving 98 patients used tactile stimulus and patient subjective assessment of sensitivity to compare a sodium citrate pluronic F 127 gel with SCH and control dentifrices. Although the pluronic gel group demonstrated the largest mean reduction in sensitivity, no significant differences were found between the groups and all three treatment groups experienced reduction in CDS.

In a subsequent eight week double-blind study involving 101 patients Collins and Perkins (1984) using subjective methodology compared two sodium citrate formulations (Protect and Protect with 0.2% NaF) with a positive control (SCH), and confirmed the previous findings. More recently McFall and Hamrick (1987) in an eight week double-blind study involving 87 patients utilised tactile stimulus and subjective cold air stimulus to compare two sodium citrate formulations, identical except for the addition of 0.1% fluoride, to one with a placebo and a 0.1% fluoride dentifrice. Both the sodium citrate formulations significantly reduced tactile sensitivity at two weeks and thermal sensitivity at eight weeks.

Other studies, however, have been inconclusive. Tarbet *et al.* (1982) in a four week double-blind study utilising electrical and cold air stimuli; together with a subjective patient assessment, compared 5% KNO_3 with SCH, sodium citrate pluronic (Protect) and formaldehyde dentifrices. Although the sodium citrate group demonstrated a decreased response the investigators concluded it was less effective than KNO_3 .

Clark *et al.* (1987) using a protocol similar to that of McFall and Hamrick (1987) failed to establish any efficacy for either the experimental dentifrice (0.2% NaF/sodium citrate buffer pluronic F 37 gel with precipitated silica abrasive) or any of the individual major ingredients.

Ong and Strahan (1989) in a six week double-blind study involving 20

patients used both objective and subjective assessment and compared a 2% dibasic sodium citrate in 'poloxamer 409' with a placebo dentifrice containing 0.76% MFP. They concluded that the sodium citrate dentifrice was not significantly more effective than the control.

Pluronic F 127 polyglycol is a surfactant with excellent wetting properties which presumably facilitates the entry of citrate ions onto the root surface (Zinner et al. 1977). Another property of the polyglycol is one of protein precipitation, and while it may be possible to decrease CDS, either by precipitating protein in the nerve receptors or mucins out of saliva which could adhere to the dentine surface and decrease tubule radii. Neither of these actions would, however, be detected using the dentine disc model (Greenhill & Pashley 1981). These investigators also suggested that the observed decrease in hydraulic conductance (L_p) was due to abrasive fillers in the dentifrice (Protect) which partially occluded the tubules. According to Zinner et al. (1977), the combined activity of the dentifrice ingredients appeared to result in the production of the citrate anion, derived from sodium citrate and citric acid, which, in conjunction with the calcium cation available in the dentinal tubules and on the tooth surface, formed a calcium citrate complex within the tubules, which presumably reduced CDS by tubule occlusion.

1.4.4.7. Calcium Hydroxide ($\text{Ca}[\text{OH}]_2$)

Calcium hydroxide ($\text{Ca}[\text{OH}]_2$) has been widely used as a cavity liner under restorations. Several investigators have also advocated its use as paste over exposed dentine (Everett et al. 1966, Jorkjend & Tronstad 1972, Levin et al. 1973, Green et al. 1977). Everett et al. (1966) suggested painting a 5% solution of $\text{Ca}[\text{OH}]_2$ on exposed cervical dentine and leaving it for one minute following a one to two minute application of sodium silicofluoride to aid precipitation of fluoride. Other investigators (Jorkjend & Tronstad 1972, Levin et al. 1973, Green et al. 1977) advocated leaving the paste for three to five minutes prior to removal of any excess, either by rinsing or cotton roll with subsequent

rinsing with sterile water. Reapplication of the paste, however, was sometimes necessary. Due to the highly alkaline nature of the paste (pH 12-14), several investigators advised that the gingival mucosa be isolated to avoid ulceration (Jorkjend & Tronstad 1972, Levin *et al.* 1973). Several studies using different methodologies have reported the effectiveness of $\text{Ca}[\text{OH}]_2$ in decreasing CDS (Jorkjend & Tronstad 1972, Levin *et al.* 1973, Green *et al.* 1977). Addy *et al.* (1983), however, questioned its reliability. Jorkjend and Tronstad (1972) reported that following periodontal surgery on 10 patients, an application of $\text{Ca}[\text{OH}]_2$ paste, which was subsequently covered with a layer of methacrylate and a periodontal pack and left for four to seven days, resulted in teeth no longer being sensitive to thermal, mechanical and sweet stimuli for up to six months post-treatment. No statistical data, however, were presented and there are problems in assessing the results of a study involving small numbers of patients. Further, since the teeth involved were subject to periodontal surgery, it is probable that any improvement in CDS was due, not to the action of the paste, but rather to natural desensitization over time.

Levin *et al.* (1973) using thermal and mechanical stimuli; together with patient subjective response, compared $\text{Ca}[\text{OH}]_2$ with magnesium hydroxide and placebo (distilled water) in 110 patients. They reported that after six months the $\text{Ca}[\text{OH}]_2$ paste was more effective. Although the placebo group showed no immediate relief, nevertheless after six months, 40% of subjects claimed complete relief.

Green *et al.* (1977), using standardised thermal and mechanical stimuli, compared $\text{Ca}[\text{OH}]_2$ with KNO_3 and placebo (water) in a double-blind three month study involving 6 patients. They claimed that $\text{Ca}[\text{OH}]_2$ was consistently more effective.

Studies on human dentine have reported that mineralization occurs when it is covered with $\text{Ca}[\text{OH}]_2$ (Mjör *et al.* 1961, Mjör 1967, Mjör & Furseth 1968, Mazetti & Toledo 1971). Trowbridge *et al.* (1982) observed (in the cat) that $\text{Ca}[\text{OH}]_2$ had little or no direct effect on dentine sensory nerve activity (SNA), and its long term effectiveness was attributed to

its ability to increase peritubular dentine.

Brännström et al. (1976), however, failed to confirm these observations. SEM revealed a variable constriction of dentinal tubules in 13/20 teeth, but only to a depth of 0.1 mm. The action of $\text{Ca}[\text{OH}]_2$ would, therefore appear to be superficial and transient in nature. No difference was observed between treated and untreated dentine.

Greenhill and Pashley (1981) observed a 21% decrease in hydraulic conductance (L_p), attributed to an increased ionized calcium concentration produced by the paste in the tubule as well as an additional effect of the high alkaline pH which would tend to convert phosphate in the tubule fluid from the more soluble HPO_4^{-2} and $\text{H}_2\text{PO}_4^{-1}$ to the much less soluble tri-basic phosphate. Both these factors, according to Greenhill and Pashley, may contribute to the precipitation of calcium phosphate and subsequent tubule occlusion. McFall (1986) suggested that the application of $\text{Ca}[\text{OH}]_2$ could result in tubule occlusion by the calcium ion tying up loose protein radicals.

Several investigators have suggested that $\text{Ca}[\text{OH}]_2$ irritates odontoblasts, stimulating them to produce secondary dentine, which would presumably result in decreased fluid flow and thereby subsequent reduction in CDS (Jorkjend & Tronstad 1972, Levin et al. 1973, Greenhill & Pashley 1981).

1.4.4.8. Potassium nitrate (KNO_3)

Hodosh (1974) reported on the effectiveness of KNO_3 as a desensitizing agent. This report was based on little more than clinical impression. Other investigators have reported on a 5% KNO_3 dentifrice with both positive and negative findings (Green et al. 1977, Tarbet et al. 1980, 1982, Collins et al. 1984, Manochehr-Pour et al. 1984, Silverman 1986). Green et al. (1977) using mechanical and thermal stimuli compared $\text{Ca}[\text{OH}]_2$ with KNO_3 and a placebo (water) in a double-blind three month study involving 6 patients. They reported that $\text{Ca}[\text{OH}]_2$ provided immediate relief from both mechanical and thermal stimuli, whereas KNO_3 only provided immediate relief from mechanical stimuli. $\text{Ca}[\text{OH}]_2$ was

considered to be more consistently effective. The KNO_3 dentifrice compared to placebo was more effective in immediately decreasing sensitivity to mechanical stimuli, but no statistically significant difference was reported in response to thermal stimuli. After three months, teeth treated with KNO_3 showed a significant decrease in sensitivity to mechanical and heat stimuli but not to cold.

Tarbet *et al.* (1980), using electrical and thermal stimuli, evaluated a 5% KNO_3 dentifrice compared to placebo silica dentifrice in a four week double-blind study involving 27 patients. A statistically significant difference for the KNO_3 dentifrice group in response to electrical stimulus was noted after two weeks of use. No statistical difference was noted (at week 4) to cold stimuli. Patients in the placebo group also demonstrated improvement to both electrical and thermal stimuli. In a subsequent four week study comparing KNO_3 (Denquel), SCH (Sensodyne), sodium citrate pluronic gel (Protect) and formalin (Thermodent) dentifrices, Tarbet *et al.* (1982) reported that KNO_3 was more effective. This study, however, did not include a true placebo (Kanapka 1990). The choice of an electrical stimulus has been criticised on the basis that it does not represent a natural stimulus normally encountered by patients suffering from CDS (**section 1.3.4.**).

Collins *et al.* (1984) in a twelve week double-blind study involving 75 patients, and using tactile and thermal stimuli together with a subjective response based on a 0-3 sensitivity scale, demonstrated that both KNO_3 and SCH dentifrices were effective in reducing CDS (**section 1.4.3.4.**). One of the problems, however, with this study is the involvement of 39 patients who had recently had periodontal surgery. As placebo and associated non-placebo effects occur during clinical trials of this nature; once again it would be difficult to differentiate the effects of natural desensitization over time following periodontal surgery (Penney & Karlsson 1976) from any significant reduction attributable to either dentifrice.

Silverman (1986) compared KNO_3 and SCH dentifrices in a twenty two week double-blind study involving 22 patients, using quantifiable tactile

(Yeaple probe) and thermal (air temperature Yeh device) and concluded that the dentifrices were equally effective. Manochehr-Pour et al. (1984), however, in a twelve week double-blind study involving 75 patients failed to demonstrate any statistically significant differences between two KNO_3 dentifrices and a placebo in their subjective response to tactile and thermally applied stimuli. Silverman (1985) compared a 5% KNO_3 /0.76% MFP dentifrice (Promise) with a KNO_3 dentifrice (Denquel) and a placebo in a twelve week double-blind clinical trial involving 68 patients. Both tactile (Yeaple probe) and thermal (dental air syringe) stimuli were used, together with a subjective patient assessment based on a 0-3 discomfort score scale. Both KNO_3 containing dentifrices, with or without MFP, were significantly more effective than placebo. As with other clinical trials which have evaluated KNO_3 , reduction in sensitivity to both tactile and thermal stimuli was evident by the second week and continued to be observed throughout the trial.

Silverman et al. (1988), using quantifiable tactile (Yeaple probe) and thermal (variable air temperature Yeh device) stimuli compared two KNO_3 dentifrices with or without 0.76% MFP, a dentifrice containing 0.76% MFP and a placebo in a twelve week double-blind clinical trial involving 60 patients. Both KNO_3 dentifrices significantly reduced CDS compared to the placebo and MFP preparations. The investigators concluded that MFP neither enhanced nor detracted from the desensitizing efficacy of KNO_3 , and that by itself 0.76% MFP was ineffective.

Person et al. (1989) using thermal (Temptronic air device) evaluated five dentifrices including 5% KNO_3 /0.76% MFP and SCH and one mouthrinse in an eight week double-blind trial involving 119 patients. KNO_3 /MFP and SCH dentifrices exhibited an improvement in warm/hot air thresholds after eight weeks of use. No statistically significant differences, however, were observed between groups.

Recently Reinhart et al. (1990) compared a glycerin gel with or without 10% KNO_3 , against a control paste in a four week pilot study involving

12 patients (36 teeth in total). The investigators concluded that the KNO_3 glycerin based gel decreased sensitivity to thermally applied stimuli (cold water 20°C , 10°C , 0°C) but this was only statistically significant at week 2, whereas the plain glycerin gel group demonstrated a statistically sustained reduction at weeks 3-4. The potential efficacy of glycerin as a desensitizing agent has been suggested by other investigators (**section 1.4.4.1.**).

Chesters et al. (1992) using electrical, tactile and thermal stimuli compared two MFP dentifrices containing either 2% potassium citrate ($\text{K}_3\text{C}_6\text{H}_5\text{O}_2$) or 2% KNO_3 , with a control dentifrice containing MFP only, in an eight week double-blind clinical study involving 120 patients. All three groups demonstrated a statistically significant reduction in CDS over the eight weeks of dentifrice use. $\text{K}_3\text{C}_6\text{H}_5\text{O}_2$ was shown to significantly reduce CDS relative to both the MFP control and KNO_3 . The investigators postulated that a possible explanation as to why the KNO_3 failed to demonstrate any benefit relative to the control was the inclusion of MFP in the KNO_3 dentifrice. Silverman et al. (1988), however, concluded that MFP neither enhanced or detracted from the desensitizing effects of KNO_3 .

Although numerous clinical studies appear to demonstrate that KNO_3 is an effective desensitizing agent, its exact mode of action of is unclear. Hodosh (1974) postulated that desensitization resulted either from its oxidizing nature, or through tubule occlusion by a crystallization process, or both, although he did not provide any evidence for this proposal.

Various in vitro studies, however, have failed to demonstrate any uptake of KNO_3 onto or into dentine (Addy & Mostafa 1988, 1989). Greenhill and Pashley (1981) and Pashley et al. (1984c) were also unable to demonstrate the effectiveness of KNO_3 , (either in a 30% solution or 5% dentifrice form) in terms of decreased fluid flow across dentine in the dentine disc model. The results of such in vitro studies would suggest that KNO_3 does not reduce CDS by tubule occlusion.

Several investigators (Kim 1986a, Markowitz & Kim 1985, 1990,

Markowitz *et al.* (1991) have demonstrated in the animal model that the important moiety of KNO_3 was the potassium salt and not the NO_3^- anion. While various divalent cation solutions were effective in reducing both INA and SNA, they were less effective than potassium which appeared to be an effective desensitizing agent regardless of the anion with which it was combined. According to Kim and co-workers (1985, 1986, 1987, 1988, 1989, 1991) the mode of action for potassium desensitization is through raising the intratubular K^+ concentration which renders the intradental nerves less excitable to further stimuli by depolarizing the nerve fibre(s) membrane. Initially this increase in the K^+ content elicits an increased number of action potentials, after the initial depolarization, however, the nerve fibre(s) cannot repolarize due to the maintained high levels of extracellular K^+ and consequently a sustained depolarized state occurs (axonal accommodation). Whether Kim's hypothesis can be extrapolated to explain how potassium containing salts exert this effect in man (e.g., from the external dentine to the inner dentine/pulp region in sufficient concentration) is debatable (Sena 1990, Orchardson & Lucas 1991, Vongsavan & Matthews 1991, 1992a,b).

1.4.4.9. Potassium chloride (KCl)

Salvato *et al.* (1989) using tactile (Yeaple probe) and thermal (cold air) stimuli; together with patient subjective assessment, compared a KCl/MFP dentifrice against a placebo dentifrice in a twelve week double-blind clinical study involving 40 patients and concluded that KCl/MFP was effective.

More recently Sidi *et al.* (1991) using a similar methodology range compared a KCl/NaF/silicon dioxide dentifrice with a commercially available dentifrice containing KCl/MFP/dicalcium phosphate (DCP) in an eight week double-blind clinical study involving 39 patients. They suggested that the KCl/NaF dentifrice with a silica abrasive may provide greater reduction in tactile sensitivity after eight weeks controlled use than a KCl dentifrice containing MFP/DCPs. Both

dentifrices, however, appeared to be comparable for the other variables measured. KCl and various cations have also been shown to decrease INA and SNA in the animal model (Markowitz & Kim 1985, Bilotto et al. 1986, Markowitz et al. 1991) (See above).

According to these investigators potassium salts may reduce CDS via an alternative mechanism of action (direct ionic diffusion) in which pulpal nerve activity (PNA) is blocked by alteration of SNA rather than by tubule occlusion although this hypothesis has been questioned (Sena 1990, Orchardson & Lucas 1991, Vongsavan & Matthews 1991, 1992a,b). (section 1.2.7.).

1.4.4.10. Fluoride

Fluoride has been used in dentifrices, gels, mouthrinses and varnishes in the treatment of CDS.

A number of studies have demonstrated the clinical effectiveness of fluoride (with or without iontophoresis) in reducing sensitivity to mechanical, thermal and/or chemically applied stimuli (Lukomsky 1941, Hoyt & Bibby 1943, Clement 1947, Jensen 1964, Everett et al. 1966, Bolden et al. 1968, Hazen et al. 1968, Kanouse & Ash 1969, Miller et al. 1969, Hernandez et al. 1972, Murthy et al. 1973, Minkov et al. 1975, Gangarosa & Park 1978, Gangarosa et al. 1978, 1989, Gangarosa 1981, Squillaro et al. 1981, Carlo et al. 1982, Thrash et al. 1983, Lutins et al. 1984, Gangarosa & McRae 1985, Klaus & Gangarosa 1986, Fukumoto et al. 1987, Kern et al. 1989, Lee et al. 1991, McBride et al. 1991).

1.4.4.11. Sodium fluoride

While the exact mechanism of fluoride in reducing CDS is uncertain, some have postulated that fluoride occluded tubules (Lukomsky 1941, Gedalia et al. 1971, Erhlich et al. 1975, Tal et al. 1976, Laufer et al. 1981, Greenhill & Pashley 1981, Pashley 1985a, Gangarosa et al. 1985, Krauser 1986, McFall 1986, Kern et al. 1989). A recent in vitro study (Addy & Mostafa 1988), however, suggested that, in common with strontium, potassium and possible zinc salts, fluoride did not appear

to produce direct permanent tubule occlusion. These investigators postulated that while an initial precipitation or reaction with the dentine surface produced a fine granular deposit, this appeared to readily dissolve in an aqueous environment. Indirect effects mediated by salivary components, however, may explain the apparent efficacy of fluoride and other compounds used to decrease CDS (Addy & Dowell 1983).

Other, SEM investigations have demonstrated granular precipitations and calcospherites, presumably composed of calcium fluoride, within dentinal tubules after topical fluoride application (Brännström & Garberoglio 1972, Samara-Wickman et al. 1972, Erhlich et al. 1975, Brännström et al. 1976, Tal et al. 1976, Penney & Karlsson 1976, Greenhill & Pashley 1981, Laufer et al. 1981), although Brännström et al. (1976) reported that following a one minute application of a cavity liner containing 3% NaF, no indications of obliteration of the dentinal tubules could be detected.

Lukomsky (1941) was the first investigator to propose the use of NaF as a desensitizing agent to treat CDS. Hoyt and Bibby (1943) reported that a paste consisting of equal parts of sodium fluoride (33% NaF), kaolin and glycerin was effective. Others have also demonstrated the effectiveness of NaF in this form (Clement 1947, Everett et al. 1966). Tarbet et al. (1979) in a four week double-blind study using electrical and cold air stimuli compared a 33% NaF paste with a placebo and found that initially a significant reduction was observed at three and seven days following application of NaF; but no further significant reduction was evident at ten days. On the basis of in vitro studies (Greenhill & Pashley 1981) postulated that any desensitizing effect of the paste may result from mechanical occlusion of tubules by either NaF, kaolin or both. As mentioned previously, glycerin has also been purported to have desensitizing properties (Colaneri 1952, Reinhart et al. 1990).

Other investigators, however, suggested that the most important variable in the topical application of a paste, is not the presence or absence of a specific ingredient, e.g., NaF, kaolin or glycerin, but the act of burnishing (Pashley 1984, 1985a, Pashley et al. 1984c).

Greenhill and Pashley (1981) in an in vitro study observed that acidulated NaF solution reduced hydraulic conductance (Lp) 24% compared to 2% NaF (17.7%), whereas 2% NaF application through iontophoresis reduced Lp by 33%. Ehrlich et al. (1975) evaluated 2% acidulated NaF. The teeth were extracted at post application intervals and examined for fluoride uptake. Fluoride levels from a single application of NaF were observed for up to fourteen days. This study appears to support the earlier in vivo and in vitro monkey and human studies which reported that teeth immersed in concentrated fluoride solution demonstrated a high fluoride uptake in surface and subsurface layers (Gedalia et al. 1971, 1977, Shulman et al. 1968, 1973). Gedalia et al. (1978) compared the effectiveness of topically applied 2% NaF with and without iontophoresis with 10% SCH, and reported that a significant reduction occurred with NaF regardless of SCH pre-treatment.

Addy et al. (1987b), however, demonstrated no benefits of a fluoride and strontium dentifrice when compared with a strontium only dentifrice.

Squillaro et al. (1981) compared 1200 ppm NaF with a placebo in a thirty day double-blind clinical study and concluded that NaF was significantly more effective than placebo, although the placebo also produced positive results. Javid et al. (1987) compared cyanoacrylate with 33% NaF using cold air stimuli during a six week clinical study. Cyanoacrylate was more effective. One of the problems in this study, however, was the lack of a suitable control as well as procedural differences when compared to other studies. 33% NaF was applied for 30 seconds on test teeth at each visit and the procedure was repeated weekly for a total of six fluoride applications. The cyanoacrylate group received a single application of cyanoacrylate at baseline. Both groups returned twenty four hours later and then at weekly intervals. A placebo effect cannot be ruled out.

Recently Thrash et al. (1992) reported on a study in which 30 patients were divided into three treatment groups (0.717% solution of F⁻, 0.4% SnF₂ gel and distilled water) and evaluated using a repeatable cold

thermal stimulus over 2, 4, 8 and 16 weeks. They reported that the 0.717% solution of F^- was more effective at 2 weeks, compared to the other groups; whereas the 0.4% SnF_2 group was more effective at weeks 4-8. In a separate study a 0.717% (1.09% NaF, 0.40% SnF_2 & 0.14% hydrogen fluoride) solution of F^- was effective in reducing CDS within 15 minutes of application, when assessed with a repeatable cold thermal stimulus.

1.4.4.12. Sodium silicofluoride

Several investigators have reported that sodium silico-fluoride in aqueous solution (0.7-0.9%) compared favourably with 2% NaF solution (Massler 1955, Stout 1955). Bhatia (1953), stated that saturated (0.6%) sodium silicofluoride for five minutes was more effective than 2% NaF solution. It has been postulated that silicic acid forms a gel with tooth calcium to produce an insulating barrier (Everett et al. 1966).

1.4.4.13. Fluoride Iontophoresis

Iontophoresis may be defined as a method using electrical potential (gradient) to facilitate uptake of ions into soft or hard tissues of the body for therapeutic purposes (Walton et al. 1979, Pashley 1985a).

Iontophoresis of fluoride for the treatment of CDS has been controversial. Several investigators (Murthy et al. 1973, Gangarosa & Park 1978, Gangarosa et al. 1978, 1989, Gangarosa 1981, Carlo et al. 1982, Lutins et al. 1984, Gangarosa & Mcrae 1985, Klaus & Gangarosa 1986, Fukumoto et al. 1987, Kern et al. 1989, Lee et al. 1991, McBride et al. 1991) have reported successful desensitization by this method, while others (Schaeffer et al. 1971, Minkov et al. 1975, Brough et al. 1985) have reported conflicting results attributable to error or to a lack of standardisation in the technique of iontophoresis used (Gangarosa & Park 1978, Gangarosa 1986).

Several studies have demonstrated that radioactive ions, including those of iodide, calcium and sodium, can penetrate dentine by iontophoresis (Sausen 1955, Stowell et al. 1961, Pashley et al. 1978a).

Ehrlich et al. (1975) also observed an increased fluoride uptake in exposed root dentine following topical application of 2% NaF, and that the fluoride ion has a marked affinity for calcium, which in turn may react in fluids to form CaF_2 . Zadok et al. (1976) demonstrated that fluoride iontophoresis when compared to topical fluoride alone resulted in an increase in fluoride ion uptake without adverse pulpal changes.

Johnson et al. (1982) in a twelve week study compared an electro-ionizing toothbrush with and without a battery and using 0.4% SnF_2 or SCH as active agents. All three groups demonstrated improvement by week 4, but no significant difference was noted until week 12, at which time both SCH without battery and 0.4% SnF_2 with battery were significantly superior to 0.4% SnF_2 without battery.

The exact mechanism of fluoride iontophoresis is not known, although several hypotheses have been proposed. Lefkowitz (1962), Scott 1962, Lefkowitz et al. (1963) and Murthy et al. (1973) suggested that the desensitization was the result of secondary dentine formation by the electrical current (iontophoresis). Gangarosa and Park (1978) proposed that iontophoresis produced paraesthesia by altering the sensory mechanism. A third possible mechanism, based on the hydrodynamic theory (Brännström 1962, 1963a,b, Brännström & Åstrom 1972) hypothesized that fluoride iontophoresis may increase the concentration and depth of penetration of fluoride ions in dentinal tubules, which in turn may cause a micro precipitation of CaF_2 , thereby occluding the tubules and reducing conduction of hydrodynamically mediated stimuli (Pashley 1985a, Kern et al. 1989).

Most studies on NaF with iontophoresis report good short-term results, although only a few have reported long-term results (Klaus & Gangarosa 1986, Gangarosa et al. 1989, Kern et al. 1989). It is important to point out, however, that neither the safety nor the efficacy of this procedure has been proved (American Dental Association 1979). Further evaluation of fluoride iontophoresis over time using adequate controls and suitable test methods, which are both quantifiable and reproducible, as recommended by the American Dental Association, Council on Dental

Therapeutics (1986) are required.

(see Appendices for a more detailed review on iontophoresis)

1.4.4.14. Stannous fluoride

Stannous fluoride (SnF_2) in a 0.4% glycerin gel has been reported to be effective in reducing CDS.

Miller *et al.* (1969) in a one week double-blind clinical study using thermal, chemical and tactile stimuli reported that when compared to placebo SnF_2 in a 0.4% glycerin gel with no abrasive component significantly reduced CDS although it should be noted that the investigation lacked an adequate pain response grading system which is necessary to quantify the effectiveness of a particular desensitizing agent (Blong *et al.* 1985). Thrash *et al.* (1983) reported that topical 0.717% aqueous SnF_2 solution demonstrated immediate reduction in CDS.

Blong *et al.* (1985) in an eight week double-blind clinical study utilising electrical and thermal stimuli confirmed the previous work of Miller *et al.* (1969). Other studies have reported negative or inconclusive findings (Bolden *et al.* 1968, Hazen *et al.* 1968, Zinner *et al.* 1977). Both the Bolden and Hazen studies evaluated 0.4% SnF_2 with 0.76% MFP, 1.4% formalin and a control dentifrice with no MFP (non MFP) in a four week double-blind study, and concluded that 0.4% SnF_2 was less effective than MFP. Bolden *et al.* (1968) also reported that 0.4% SnF_2 was less effective than either the formalin or control dentifrices, while Zinner *et al.* (1977) reported 0.4% SnF_2 did not show any significant difference compared with the control group using a non-fluoride dentifrice. Several studies have also utilised various iontophoresis devices to compare SnF_2 with a placebo (Schaeffer *et al.* 1971, Johnson *et al.* 1982). Schaeffer *et al.* (1971) compared SnF_2 with a placebo and a control group utilising an iontophoretic toothbrush and reported no significant difference over placebo.

To date, evidence as to the efficacy of 0.4% SnF_2 gels is inconclusive. It is noteworthy, however, that the American Dental Association Council on Dental Therapeutics has not accepted them for their effect on CDS,

plaque or gingival and periodontal state (Hastreiter 1989).

Recently Barbakow et al. (1992) reported that a precipitate free SnF_2 -Amine fluoride gel containing 10,000 ppm F^- provided optimal protection to dentine in vitro against acid etch and induced a better F^- retention in dentine, compared to similar gels with lower F^- concentrations.

Several in vitro investigations have demonstrated that SnF_2 may precipitate on the dentinal surface and occlude tubules (Blunden et al. 1981, Dowell & Addy 1984, Ellinsen & Rolla 1987, Addy & Mostafa 1988, 1989). Penney and Karlsson (1976) reported in the dog model, following either pumicing or pumicing and etching, that both Sn and fluoride penetrated deep into the dentine with fluoride penetrating at least twice the distance of Sn regardless of the pre-treatment condition.

Scott (1982) and Krauser (1986) have postulated that the mode of action of SnF_2 is through calcific blockage of the dentinal tubule.

Addy and Mostafa (1988) on the basis of their in vitro studies reported that tin salts, rather than fluorides of the other salts tested, appear to have the potential to directly occlude tubules and suggested that this could be the mechanism by which clinical efficacy has been achieved, as reported by earlier investigators (Miller et al. 1969, Johnson et al. 1982, Thrash et al. 1983, Blong et al. 1985).

SnF_2 when placed in an aqueous environment appears to undergo hydrolysis and precipitates out of solution. Miller et al. (1969) resolved this problem by placing SnF_2 in solution in glycerin without the use of water.

1.4.4.15. Sodium Monofluorophosphate

Sodium Monofluorophosphate (MFP) has been demonstrated to be an effective anti-carries agent in younger populations (Ripa 1989, Mellberg 1991). Several investigators have also demonstrated the effectiveness of 0.76% MFP in the management of CDS (Bolden et al. 1968, Hazen et al. 1968, Kanouse & Ash 1969, Hernandez et al. 1972, Perminder et al. 1985), although Silverman et al. (1988) reported that by itself 0.76% MFP in a

dentifrice is ineffective. Bolden et al. (1968) using a tactile stimulus compared a dentifrice containing 0.76% MFP with three other dentifrices, a non-MFP control, 1.4% formalin, and 0.4% SnF₂ in a four week double-blind study involving 115 patients. The investigators reported a 66% improvement with the 0.76% MFP dentifrice; although even the non-MFP control demonstrated a 46% improvement.

A similar study by Hazen et al. (1968) reported a 58.5% improvement compared to the non-MFP control which demonstrated a 38% improvement. The 0.4% SnF₂ dentifrice demonstrated a 55% improvement, compared with 41.9% in the Bolden study; while the 1.4% formalin dentifrice demonstrated less improvement (33.8%) compared to the non-MFP control.

Kanouse and Ash (1969) using a calibrated thermoelectric device (Smith & Ash 1964a,b) compared 0.76% MFP with a non-MFP placebo in a three month double-blind clinical study involving 59 patients. Patients using MFP had an increased tolerance to cold and hot stimuli compared to the non-MFP placebo. These investigators concluded that the results demonstrated the effectiveness of 0.76% MFP in reducing CDS.

Hernandez et al. (1972) using a tactile stimulus compared 0.76% MFP, SCH, and non-MFP control dentifrices in a twelve week cross over study involving 276 patients. After six weeks of assigned dentifrice use, both 0.76% MFP and SCH dentifrices demonstrated statistically significant reductions in CDS; 61.3% and 37.8% respectively, as compared with the non-MFP control (19.5%). 0.76% MFP, however, was significantly better than SCH. After a further six weeks using a non-MFP control, the original control group demonstrated a 41.4% reduction; whereas the original 0.76% MFP and SCH groups continued to maintain their desensitizing effect (51.3% and 35.3% respectively). The results indicated that the desensitizing effect provided by the 0.76% MFP and SCH dentifrices following the cessation of six weeks dentifrice use, lasted for at least a further six weeks.

Shapiro et al. (1970a,b), however, failed to demonstrate any statistically significant difference between SCH and 0.76% MFP dentifrices in an eight week clinical study (**section 1.4.3.4.**).

Perminder et al. (1985) using two standardised thermal devices for measuring hot and cold stimuli compared 0.76% MFP, non-MFP placebo and 1.3% formalin containing dentifrices in a six week double-blind clinical study involving 90 patients. There was no significant difference between the 0.76% MFP and 1.3% formalin dentifrices to hot stimuli; whereas for the cold response, the 0.76% MFP dentifrice was significantly different to 1.3% formalin. As with the previously mentioned studies the non-MFP placebo also provided a beneficial reduction in CDS.

Other investigators have evaluated MFP incorporated with other desensitizing agents in dentifrices; for example 0.8% MFP with 1.3% formalin (McFall & Morgan 1985), 5% KNO_3 and 0.76% MFP (Silverman 1985, Silverman et al. 1988, Person et al. 1989), 2% KNO_3 or $\text{K}_3\text{C}_6\text{H}_5\text{O}_2$ and MFP (Chesters et al. 1992), KCl and MFP (Salvato et al. 1989, Sidi et al. 1991). Interpretation of the effectiveness of MFP incorporated with other desensitizing agents varies. Chesters et al. (1992) postulated that a KNO_3 dentifrice failed to demonstrate any benefit relative to a control because of MFP in the KNO_3 dentifrice. Silverman et al. (1988), however, concluded that MFP neither enhanced nor detracted from the desensitizing effects of KNO_3 , but that by itself 0.76% MFP in dentifrice form is ineffective in reducing CDS.

The mechanism of action of MFP is unclear (Addy & Dowell 1983) although Gron and Caslavaska (1981) have demonstrated that in enamel MFP can be hydrolysed at the surface of the apatite crystals and the fluoride subsequently incorporated into the hydroxyapatite lattice. No direct evidence for such an interaction has been demonstrated in dentine (McFall 1986).

Blunden et al. (1981) reported in an SEM study that MFP produced no observed changes on the dentine surface and the tubules remained patent. Addy and Morgan (1982) in an in vitro SEM study reported that when applied to dentine MFP increased electrical resistance and, while the dentinal tubules appeared to be occluded, the investigators postulated that this may have been due to other ingredients within the

dentifrice. Such tubule occlusion, however, does not appear to be permanent and, in common with other fluorides, strontium, potassium and possibly zinc salts, the precipitant was readily dissolved in an aqueous environment. As such, MFP does not appear to produce permanent tubule occlusion (Addy & Mostafa 1988).

1.4.4.16. Nicomethanol hydrofluoride

Nicomethanol hydrofluoride is a relatively new amine fluoride which has been incorporated in a dentifrice for the prevention of caries. In vitro studies on human teeth demonstrated that nicomethanol hydrofluoride increased enamel fluoride concentration following brushing (Barbakow et al. 1986), as well as decreasing enamel solubility (Vezin et al. 1985). Lecointre et al. (1986) compared a nicomethanol hydrofluoride containing dentifrice with one containing KNO₃/MFP in a four week double-blind clinical study involving 76 patients with CDS. They concluded that the nicomethanol hydrofluoride dentifrice was at least as effective on the basis of responses to tactile (probe) and thermal stimuli (cold air) and patient subjective response.

The problems of evaluating the efficacy of a desensitizing dentifrice using these methods of assessment have been discussed elsewhere (section 1.3.).

The exact mode of action is unclear, but it may be similar to that of other fluoride compounds which appear to decrease CDS by tubule occlusion (Lukomsky 1941, Gedalia et al. 1971, Ehrlich et al. 1975, Tal et al. 1976, Greenhill & Pashley 1981, Laufer et al. 1981). Further, availability of fluoride in a desensitizing dentifrice may help to prevent root caries in an adult population (Jensen & Kohout 1988).

Further studies, however, are required using accepted test methodology to ascertain the effectiveness of nicomethanol hydrofluoride as a desensitizing agent.

1.4.4.17. Remineralising mouthwash

A recent in vitro study (Lussi et al. 1989) evaluated the influence of

different fluoride treatments on erosion lesions in a pH cycling experiment. All groups with 3ppm fluoride in a remineralising solution containing 2mM Ca^{2+} and 3.4mM PO_4^{3-} concentrations showed greater remineralisation rates; although the erosion rates were significantly smaller with topical fluoride than fluoride in the remineralisation solution. 3ppm amine fluoride (Elmex 335) in remineralisation solution, however, yielded a significantly smaller erosion rate during the five day experimental period than the other treatments.

Clark et al. (1990b) and Ito et al. (1991) evaluated a remineralisation solution containing Ca^{2+} , PO_4^{3-} and fluoride in a double-blind clinical study involving 27 and 34 patients respectively. Patients rinsed twice daily with either the remineralising solution or a placebo rinse. Evaluation was by mechanical (Yeaple probe) and thermal (0.05ml of 5°C water) stimuli; together with a patient subjective response using a (0-8) VAS scale. The remineralising solution significantly reduced CDS due to toothbrushing and cold water stimuli, compared to placebo. Ito et al. (1991) used a replica impression technique to evaluate a remineralisation solution. Observation of the epoxy resin replicas of study teeth taken at screening and at final examination demonstrated that the remineralising solution covered the original dentine surface with an extrinsic layer, whereas there was little or no morphological change following placebo treatment.

Although both these studies reported that the remineralisation solution reduced CDS compared to placebo, more long term information is required before this particular desensitizing agent can be accepted. Criticism can also be made of the thermal stimuli (0.05ml of 5°C water) used in the Clark et al. (1990b) study (**section 1.3.3.**). It is also unclear, exactly how long the study lasted.

From this review there is some evidence to suggest that fluoride in its various formulations can reduce CDS (Addy & Dowell 1983). It should be noted, however, that CDS is still prevalent in societies where the use of fluoride dentifrices is widespread (Krauser 1986, Addy & Mostafa 1988, Kanapka 1990).

The availability of fluoride in a desensitizing dentifrice, however, may be of greater importance for caries prevention.

1.4.4.18. Potassium oxalate

Recently there has been renewed interest in the ability of oxalate salts (Potassium, Ferric & Aluminium) to reduce CDS through tubular occlusion.

Potassium oxalate ($K_2C_2O_4$) has been shown to reduce hydraulic conductance (Lp) or fluid flow in vitro in the dentine disc model (Greenhill & Pashley 1981, Pashley et al. 1978b, 1984c, 1987, Pashley & Galloway 1985a, Kaminske et al. 1990), although clinical results have been less convincing and more difficult to interpret due to the different methodologies employed, the relatively small sample size and the limited duration of the studies (Smith et al. 1988, Muzzin & Johnson 1989, Cooley & Sandoval 1989, Seo & Park 1991).

Further studies utilising the same in vitro model have demonstrated that calcium oxalate crystals precipitate onto the dentine surface and occlude tubule orifices (Greenhill & Pashley 1981, Pashley et al. 1978b, 1984c, 1987, Pashley & Galloway 1985a, Kaminski et al. 1990).

Pashley et al. (1984c) compared the effects of various desensitizing dentifrices and placebos without the active ingredient on dentine permeability and concluded that only 2% $K_2C_2O_4$ at all dilutions (1:3, 1:1, 3:1) produced a statistically significant reduction in hydraulic conduction (Lp).

Kaminski et al. (1990) compared acidic $K_2C_2O_4$ (Protect) and calcium phosphate on dentine permeability through tubule occlusion in the dentine disc model. Both solutions were applied for four minutes, calcium phosphate in a two step procedure with three minutes of calcium nitrate solution followed by a one minute of potassium phosphate. $K_2C_2O_4$ was applied for four minutes. Both treatments reduced hydraulic conductance (Lp), but only $K_2C_2O_4$ withstood an acid etch challenge. SEM demonstrated that following calcium phosphate application, the tubules were covered with calcium phosphate precipitate; whereas following

$K_2C_2O_4$ the tubules remained visible, suggesting dissolution of dentine followed by reprecipitation within the tubules.

Other in vitro dentine permeability studies have confirmed that $K_2C_2O_4$ can reverse the effect of acid etching (Pashley et al. 1978b, 1984c, 1987, Pashley & Galloway 1985a,c). Pashley et al. (1987) compared burnishing alone, kaolin in glycerin, NaCl in glycerin, NaF in glycerin, the complete NaF/kaolin/glycerin paste and topical 3% half-neutralised oxalic acid (positive control). Topical 3% half-neutralised oxalic acid was applied to the dentine disc for two minutes without burnishing and produced a surface resistant to acid etching. Pashley and Galloway (1985a,c) reported that oxalate salts decreased the permeability of the smear layer. The greatest reduction in hydraulic conductance (L_p) prior to and after acid etching was observed following sequential topical application of 30% dipotassium oxalate for two minutes followed by 3% acidic monopotassium-monohydrate oxalate for a further two minutes. The SEM appearance of the dentine smear layer revealed a heterogeneous mixture of crystals which completely obscured the surface. Following exposure to acid for two minutes, there was little change in appearance apart from a slight rounding of the crystals unlike the KCl treated dentine which was devoid of a smear layer and demonstrated visible tubule orifices. Gao et al. (1991) in an in vitro study compared the effects of topical and iontophoretic application of 2% NaF, 30% $K_2C_2O_4$ /3% monohydrogen-monopotassium oxalate on dentine permeability. Two percent NaF had no effect on $K_2C_2O_4$ induced dentine permeability reduction immediately following treatment or one week later. Although permeability reduction was decreased by 15% in the iontophoretic treatment, this was not significant. These investigators reported that permeability reduction by iontophoresis had a longer lasting effect.

On the basis of their SEM observations, Pashley and Galloway (1985a) postulated that oxalate reacted with calcium ions within the dentinal fluid to form insoluble calcium oxalate crystals, which subsequently blocked the tubule orifices.

Application of 30% $K_2C_2O_4$ (pH 5.6) produced larger crystals 1-2 μm in diameter (Greenhill & Pashley 1981) of calcium oxalate dihydrate which appeared to occlude relatively large tubule orifices and not the narrower, partly occluded tubules. Three percent monopotassium-monohydrogen oxalate (pH 2.0) reacted with the calcium ions to form smaller sized crystals (0.05 μm in diameter), probably a mixture of calcium phosphate and calcium oxalates capable of occluding the narrower tubule orifices.

Hirvonen et al. (1984) studied the effect of acid etching, $K_2C_2O_4$ and resin impregnation of dentine on nerve responses to dentinal stimulation (e.g., probing and air blasts) in the dog model. They reported that responses were either greatly diminished or absent following a two minute application of $K_2C_2O_4$ on acid etched dentine. SEM of epoxy resin replicas and specimens of dentine demonstrated that in contrast to acid etched dentine with exposed open tubules, $K_2C_2O_4$ treated specimens showed partially or completely occluded tubules. Kim (1986b) also reported that in the animal model $K_2C_2O_4$ reduced pulpal sensory nerve activity (SNA).

Several studies have evaluated $K_2C_2O_4$ clinically. Smith et al. (1988) in a four week double-blind clinical study involving 28 patients, reported that dipotassium oxalate, significantly decreased CDS. The data are of limited value, being based on thermal stimuli i.e., water at different temperatures 30, 20, 10 and 4°C (**section 1.3.**), small sample size and short duration of study. Muzzin and Johnson (1989) in a four week double-blind clinical study involving 17 patients, utilising a similar thermal technique compared distilled water followed by 30% dipotassium oxalate, distilled water followed by 3% monohydrogen-monopotassium oxalate, 30% dipotassium oxalate followed by 3% mono-hydrogen-monopotassium oxalate, and distilled water only. They reported a decrease in CDS following 3% monohydrogen-monopotassium oxalate and the combined sequential application of 30% di-potassium oxalate followed by 3% monohydrogen-monopotassium oxalate.

Cooley and Sandoval (1989) in a three month single-blind study

involving 28 patients, utilising a thermal stimulus, together with a questionnaire with a five point rating scale for sensitivity and concluded that the combined oxalate solutions were no more effective than distilled water in reducing CDS. It is questionable whether a cup of cold water (10°C) is a reliable test for CDS assessment, since it is not limited to the cervical area of the sensitive tooth in question and may elicit discomfort from other areas, e.g., pulpal discomfort from faulty restorations. Secondly it is not clear from the paper, whether a cold water rinse was applied pre-treatment for assessment purposes. It would appear that patients only completed a pre-test questionnaire which rated their perception of sensitivity. If so, then the pre-test values may be invalid and subsequent interpretation of the data suspect.

Both Muzzin and Johnson (1989) and Cooley and Sandoval (1989) reported an immediate decrease in CDS following application of the oxalate solutions, although contrary to Hansson (1987), no significant difference between treatments occurred at four weeks or three months.

Muzzin and Johnson (1989) also reported that Hansson did not dry the test teeth between the application of the two oxalate solutions. Cooley and Sandoval (1989) did not provide such information.

Seo and Park (1991) reported a study involving 76 patients, which compared the short term effects of $K_2C_2O_4$, NaF and a control. $K_2C_2O_4$ was reported to show the best effect.

Cuenin et al. (1991) also reported a study involving teeth scheduled for extraction in 13 patients. They reported that a low pH 3% NaCl solution was more effective than $K_2C_2O_4$ in reducing CDS. SEM observation of teeth treated by the two solutions demonstrated that both solutions reduced tubule aperture size, although the 3% NaCl solution appeared to be better. These investigators, however, did not explain how an acidic NaCl solution could reduce CDS. Markowitz & Kim 1990 have demonstrated (in a neurophysiological model) that with the application of a physiological saline solution the frequency of nerve fibre activation is very low whereas application of a hypertonic NaCl (3M)

solution produced a higher frequency of neuronal activity.

Kerns et al. (1991) in a 4 week in situ study involving 9 patients in which dentine sections of extracted teeth were either incorporated into the patients' dentures or served as controls. Each section was treated in three ways, one was root planed, one root planed and subjected to sequential $K_2C_2O_4$ application for four minutes and a final sample was etched with 0.5M EDTA for two minutes and served as a further control. In a separate study one sample was etched with 0.5M EDTA and sequential application of $K_2C_2O_4$ for two minutes. Subsequent SEM comparison of sections not incorporated into a denture demonstrated either a smooth amorphous smear layer (root planed) or a dense covering of calcium oxalate crystals ($K_2C_2O_4$) which completely obscured the underlying tubules. Sections incorporated into the denture revealed either partial smear layer dissolution and open tubules (root planed) or relatively few oxalate crystals ($K_2C_2O_4$) after 7 days. SEM observation of 0.5% EDTA samples which were adhered to resin in a patient's denture for 28 days, demonstrated a gradual occlusion of the tubules, which suggested that dentinal tubules can be occluded by the growth of crystals from salivary minerals which may be responsible in part for the observed spontaneous reduction in CDS. These investigators concluded that tubule occlusion following smear layer creation and $K_2C_2O_4$ application is relatively short lived, although such application may initially reduce CDS prior to natural occlusion of the dentinal tubules.

Two theories have been postulated to explain the mode of action of $K_2C_2O_4$ in reducing CDS. Pashley (1985a) suggested that $K_2C_2O_4$ combines the tubule-occluding properties of calcium oxalate with the inhibitory property of potassium on intradental nerves; although this alternative mechanism has been questioned (**section 1.2.7.**). Kerns et al. (1991), reported that tubule occlusion following $K_2C_2O_4$ is relatively short lived.

Although the in vitro studies suggest that $K_2C_2O_4$ would be an ideal desensitizing agent, clinical studies have been inconclusive. As with other agents there is still a need to evaluate $K_2C_2O_4$ over time using

adequate controls and suitable test methods, as recommended by the American Dental Association, Council of Dental Therapeutics (1986).

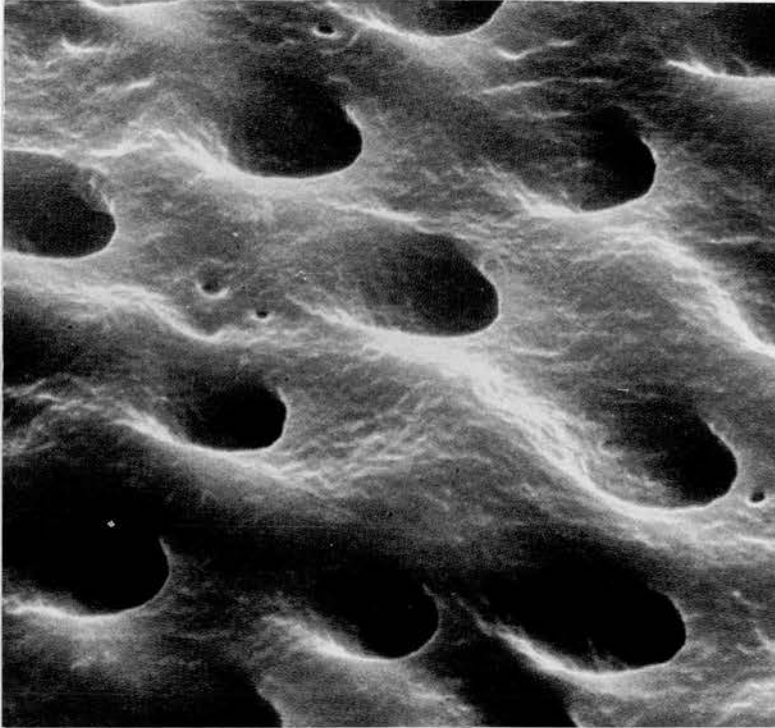
1.4.4.19. Ferric and Aluminium Oxalate

A multistep procedure for obtaining a strong adhesive bonding between tooth surface and composite resins was developed by Bowen et al. (1982). This involved sequential application of ferric oxalate [$\text{Fe}_2(\text{C}_2\text{O}_4)_3$], a 10% acetone solution of NTG-GMA (the adduct of N(p-tolyl) glycine & glycidyl methacrylate) and a 5% acetone solution of PMDM (the addition reaction product of pyromellitic dianhydride & 2 hydroxyethyl methacrylate) to the dentine or enamel followed by placement of the restorative material. The most important step in terms of dentine permeability would appear to be the application of 6.8% acidic ferric oxalate which removed the smear layer and smear plugs (Bowen et al. 1982, Bowen & Cobb 1983, Pashley et al. 1988). The resultant reaction products appears to include insoluble calcium oxalate and insoluble ferric phosphate which occlude or partially occlude the dentinal tubule orifice (Blosser & Bowen 1986, Pashley et al. 1988, Yeh et al. 1990) (Figs. 1.3.-1.4.).

Pashley et al. (1988) reported that in vitro treatment of dentine with ferric oxalate (pH 0.9) produced a significant decrease in dentine permeability (65%), whereas NTG-GMA or PMDM alone were not as effective in reducing dentine permeability (Lp). Combined NTG-GMA and PMDM increased Lp, while these agents following ferric oxalate treatment reduced Lp. Yeh et al. (1990) also reported that a one minute application of 6.8% ferric oxalate in nitric acid (Sensodyne sealant) reduced dentine permeability by 97% in the same dentine disc model. Salvato et al. (1990) in an eight week double-blind clinical study involving 38 patients, utilising tactile (Yeaple probe), thermal (cold air) and subjective methods of assessment reported that 6% ferric oxalate was effective for relief of CDS.

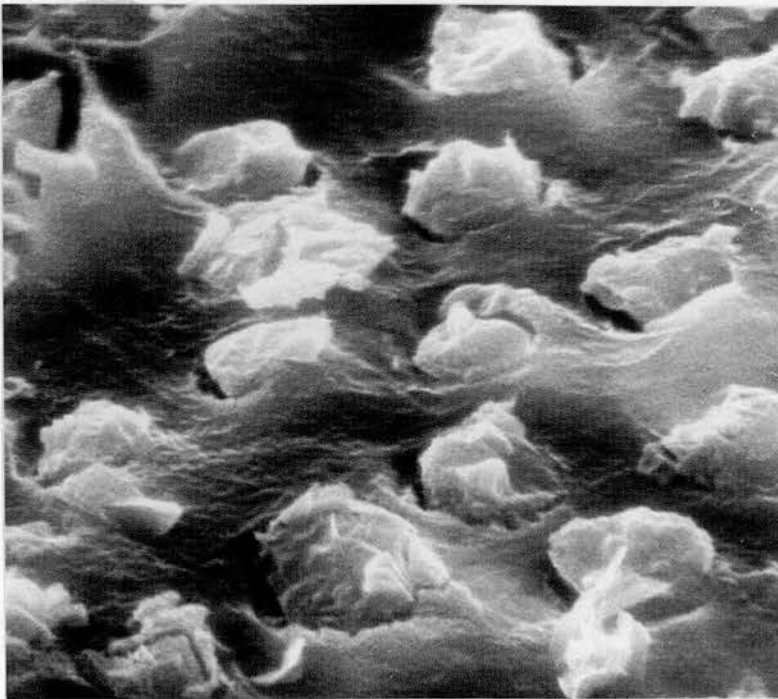
Blosser and Bowen (1988), however, demonstrated that 2.5% HNO_3 , a contaminant leftover from the synthesis of ferric oxalate, was

Figure 1.3.



Untreated dentinal tubules following acid etching
(Dentine disc)

Figure 1.4.



Dentinal tubules treated with 6.8% Ferric oxalate
Reproduced from promotional literature
(Acknowledgement Block Drug Co. Inc.)

responsible for removal of the smear layer previously associated with ferric oxalate. The application of purified ferric oxalate was ineffective in removing the smeared surface material from either enamel or dentine.

Johnson et al. (1989) reported that HNO_3 dissolved the disturbed surface layer enlarging the tubule orifices and left a largely cationic tooth surface. Ferric oxalate containing HNO_3 gave higher bond strengths than purified ferric oxalate (Blosser & Bowen 1988).

Pashley et al. (1991) compared ferric oxalate, aluminium oxalate and Tenure, a dentine conditioner containing aluminium oxalate in 2.5% HNO_3 , and reported that all acidified oxalate solutions reduced dentine permeability when applied for one minute. Treatment with Tenure dentine conditioner for 60 seconds, however, did not reduce permeability as well as the other oxalates. The observation that 2.5% HNO_3 increased permeability compared to the other oxalates appears to substantiate claims that 2.5% HNO_3 could be responsible for the initial removal of the smear layer and smear plugs rather than ferric or aluminium oxalate (Blosser & Bowen 1988, Johnson et al. 1989). They postulated that HNO_3 solution dissolved the mineral component of the smear layer within ten seconds. Insoluble salts of aluminium or ferric phosphate and calcium oxalate formed which subsequently reduced dentine permeability. Although these studies indicate that 2.5% HNO_3 may be responsible for smear layer removal, it would be somewhat premature to use HNO_3 containing no metal oxalate on vital teeth (Blosser & Bowen 1988).

Kim (1986b) reported that lithium nitrate together with aluminium and ferric oxalate compounds had varying effects on pulpal SNA in the animal model. Ferric compounds produced the greatest reduction in SNA, with increasing molar concentrations indicating a dose-related response. SNA was no longer recorded following ferric application which, according to Kim, indicated that the dentinal tubules were totally blocked. Aluminium ammonium sulphate also caused a gradual decrease in SNA with increasing molar concentrations, but this was considered unreliable, because of variability with aluminium compounds.

To date no adverse pulpal effects have been reported in bio-compatibility studies involving ferric oxalate in the multistep dentine bonding process (Siew et al.1984, Stanley et al.1985a,b, Chohayeb et al.1985, Dumsha & Beckerman 1986, Bowen et al.1987a,b, Blosser et al.1988).

One of the problems with ferric oxalate under oral conditions is that it may leach out of the precipitate layer and react with hydrogen sulphide produced by oral bacteria to form the insoluble black precipitate ferric sulphide (Asmussen & Bowen 1987). Bowen (1986) substituted 6.8% aluminium oxalate for ferric oxalate, which appeared to have little effect on the bond strengths between tooth surface and restorative material (Asmussen & Bowen 1987). This substitution also reduced the surface treatment of enamel or dentine to two rather than three steps (Bowen et al.1987a,b).

The long term solubility characteristics produced by ferric and aluminium oxalate on the modified dentine surface are not clear (Bowen et al.1987a,b, Gwinnett 1988, Pashley et al.1991).

A feature of aluminium oxalate treatment is the formation of insoluble reaction products which may obliterate the tubules (Bowen et al.1987a,b, Araujo & Asmussen 1989, Pashley et al.1991). Gwinnett (1988), however, reported that contrary to ferric oxalate, reaction products following aluminium oxalate and 2.5% HNO₃ treatment seldom occluded the tubules. This may be because obliteration decreases as pH of pre-treatment solution increases. At pH 2.0, patent tubules are observed (Araujo & Asmussen 1989). Gwinnett (1988) also reported that the pH of the aluminium oxalate solution increased from 0.75 at the commencement to approximately 4.0 at the end of treatment.

Pashley et al.(1991) postulated that following ferric and aluminium oxalate application, one would expect to observe a variety of calcium phosphate, ferric phosphate, aluminium phosphate and calcium oxalates on the modified dentine surface occluding the tubules.

On the basis of these in vitro studies, both ferric and aluminium oxalates appear to have promise as potential desensitizing agents,

although to date, only one clinical study (Salvato et al.1990) has evaluated the efficacy of ferric oxalate as a desensitizing agent in relieving CDS.

Further studies, however, are required in order to establish the long term efficacy of both ferric and aluminium oxalate in relieving CDS.

1.4.4.20. Magnesium Sulphate

Four percent magnesium sulphate applied by iontophoresis for three minutes was effective in reducing CDS (Guo-Hua & Morimoto 1991). The presence of granular deposits on the dentine surface was demonstrated by SEM. Clinically 4% magnesium sulphate relieved CDS over a 25 week period. Four percent magnesium sulphate may act both as a tubule occludent as well as by blocking nerve transmission (Xu 1980. Cited by Guo-Hua & Morimoto 1991). However, a group should have been included to ascertain the effect of iontophoreseis with or without the active agent. The study also relied on patient response to methodology of dubious objectivity (**section 1.1.3.**) as well as using words such as good, moderate and poor to describe subsequent relief from CDS. Doubts may also be expressed concerning the blindness of the study, since it would appear that patients may have been aware that 4% magnesium sulphate was applied on the right side of the jaw and 4% KNO₃ on the contralateral side, and in the case of 16 teeth rated as poor in the 4% KNO₃ group, these were subsequently retreated with 4% magnesium sulphate. It is not clear whether these teeth were subsequently excluded from analysis.

Further studies are, therefore, required using more objective methodology before 4% magnesium sulphate can be accepted as a desensitizing agent.

1.4.4.21. Tartar control dentifrices

A recent in vitro study (Mason et al.1989) reported that tartar control dentifrices reduced hydraulic conductance (Lp) in the dentine disc model. SEM demonstrated an amorphous particulate layer which

occluded exposed tubules. Tartar control dentifrices do not appear to remove the smear layer in vitro, although once the tubules were exposed following acid etching, these dentifrices reduced hydraulic conductance (Lp) through tubule occlusion.

Tartar control dentifrices have, however, been associated with CDS in vivo (Kowitz & Meng 1989). A fourteen week double-blind study involving 89 patients compared the effects of two dentifrices, one with tartar control agents and the other without, and indicated low hypersensitivity, not significantly higher than non-tartar control dentifrices. This study, however, did not utilise any recognised methodology for assessing CDS, relying only on an opinion survey based on a questionnaire taken at two weekly intervals throughout the study. It is also unclear whether those patients who experienced increasing discomfort to cold food or liquid when using tartar control dentifrices stayed in the study or dropped out.

The apparent discrepancy between the results of the in vitro and in vivo studies highlight the difficulties in assessing whether a particular active ingredient is effective when used in vivo. Logically if the tartar control dentifrice reduces Lp through tubule occlusion, then it is difficult to interpret the result of the Kowitz & Meng (1989) study in which they reported a not significantly higher level of discomfort in the tartar control group.

Further clinical studies of tartar control dentifrices, using accepted methodology to assess CDS, are required if these dentifrices are to be used to treat CDS.

1.4.4.22. The abrasive component of desensitizing dentifrices

Desensitizing dentifrice studies have usually attributed any reduction in CDS to the efficacy of the active ingredient. Several investigators, however, have proposed that other dentifrice ingredients, notably the abrasive component, may be responsible (Hiatt & Johnson 1972, Addy & Morgan 1982, Mostafa et al. 1983, Pashley et al. 1984c, Addy et al. 1987a,b, Absi et al. 1989b, Addy & Mostafa 1989).

Abrasive components, notably silica and to a lesser extent alumina, have been observed on the surface, as well as occluding the tubules, of dentine specimens which had been brushed with different dentifrice slurries (Addy & Morgan 1982, Mostafa et al.1983, Addy et al.1985, Absi et al.1989b, Addy & Mostafa 1989).

Addy and Mostafa (1989) observed that these granular deposits were almost certainly derived from dentifrice abrasive components for some dentifrices, this could be verified by the same effects being produced by the abrasive system alone.

Retention of the granular layer was readily influenced by washing. Only the fine silica-based dentifrices were little affected by washing for one hour, whereas Sensodyne with the abrasive, diatomaceous earth, was readily removed by washing. A parallel clinical study appeared to be consistent with the in vitro observations in that SrAc₂F dentifrices were more effective than Sensodyne and Emoform (Addy et al.1987b). Dentifrice abrasives may, desensitize teeth through tubular occlusion and by increasing the mechanical smearing of dentine by the toothbrush (Pashley et al.1984c, Absi et al.1989a). Apart from the abrasive component, other dentifrice ingredients may also influence the abrasivity of a dentifrice. Sodium lauryl sulphate is a surfactant widely used in dentifrices and reported to influence the abrasion process (Redmalm 1986) and also to remove the smear layer (Absi et al.1992).

Commercial and test dentifrices have used a variety of abrasive components including alumina, diatomaceous earth, dicalcium phosphate, calcium and magnesium carbonate, silica and silicon dioxide (Barbakow et al.1987a, Addy & Mostafa 1989). Abrasivity has been investigated using techniques such as weight loss, shadow graphic methods, radio-tracers, microscopy, diffusion of laser light and surface profilometry (Barbakow et al.1987b,c). Hembree & Hembree (1977) and Desautels & Labreche (1988) reported that Sensodyne, with the abrasive diatomaceous earth, produced more abrasion in the weight loss model than other dentifrices tested. Recent figures based on a modified radio-tracer

technique (Grabenstetter et al. 1958) reported that Sensodyne had a mean Radioactive Dentine Abrasion (RDA) value of 101 compared to the highest acceptable RDA value of 245 (**Fig.1.5.**). Discrepancies may therefore, be the result of the choice of test method used. Stookey and Muhler (1968) evaluated 43 commercial dentifrices using both weight loss and radio-tracer techniques. The relative abrasivity of Sensodyne was high using weight loss, whereas the radio-tracer technique value (284) placed the dentifrice in the intermediate abrasiveness category (200-400).

The Radioactive Dentine Abrasion (RDA) test has been recognised as a definitive quantitative measurement technique for testing dentifrice-associated dentine removal (Hefferren 1976). Hahn & Kim (1991) recently reported that the RDA and surface profile methods were more reproducible and precise than the weight loss method.

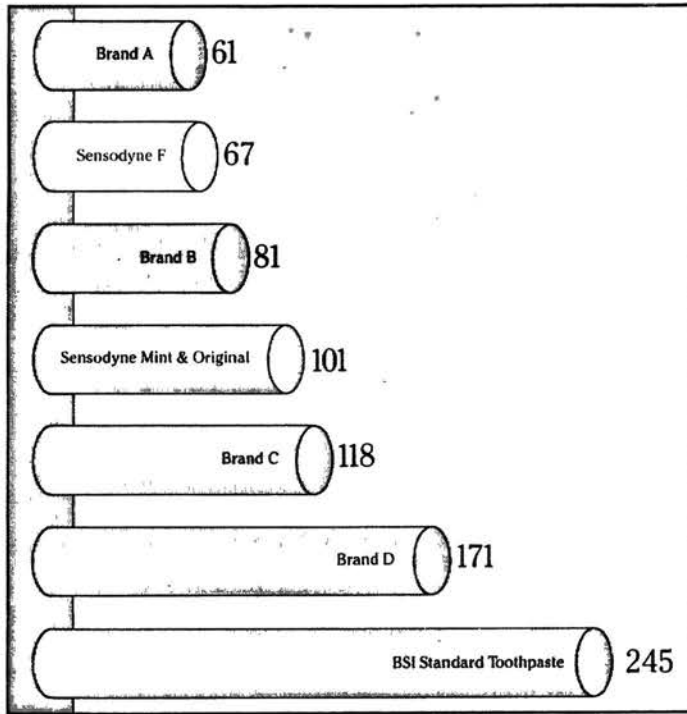
Recent studies (Manochehr-Pour et al. 1984, Silverman 1985, McFall & Hamrick 1987, Addy et al. 1987b, Salvato et al. 1989, Jackson et al. 1989, 1990, Sidi et al. 1991) have utilised a low abrasive component in desensitizing dentifrices with varying results and further studies are indicated to ascertain whether changing the abrasive component would effect any change in desensitizing efficacy.

Traditionally desensitizing agents have been evaluated using the previously described in vitro models, which have identified those agents which have the potential of occluding dentinal tubules.

An alternative method by using impression replication techniques has been reported (Hirvonen et al. 1984, Absi et al. 1987, 1989a, Lee et al. 1991). Absi et al. (1989a) used a silicone rubber impression material (Optosil) which identified sensitive and non-sensitive areas. This supported the observation (Absi et al. 1987) that hypersensitive dentine had many dentinal tubules open at the surface; whereas non-sensitive dentine had fewer and narrower open tubules. Garnick et al. (1992), however, demonstrated the limitations of the replica technique particularly in regard to cleaning the root surface with 1% NaOHCl.

Neither the Pashley dentine disc nor replication techniques take into

Figure 1.5.



Mean Radioactive Dentine Abrasion (RDA) Values of
desensitizing dentifrices from data on file
(Acknowledgement Stafford Miller Ltd.)

account possible reaction of desensitizing agents with the constituents of dentinal fluid, nor the possible in vivo impact of these agents on pulpal nerves (Absi et al. 1989a, Sena 1990).

To date, despite claims to the contrary, no desensitising agent or technique have fulfilled Grossman's (1935) requirements for an ideal desensitizing agent by reducing CDS through tubule occlusion.

1.4.5. Tubule sealants

1.4.5.1. Adhesives and Resins

Resins and adhesives probably act by sealing tubule orifices, although this impregnation may be superficial. Material can be displaced over time and sensitivity may reoccur (Nordenvall et al. 1984).

Several investigators have suggested using adhesives and resins for the treatment of CDS (Dayton et al. 1974, Brännström et al. 1979, Wycoff 1982, Nordenvall et al. 1984, Fusayama 1988) by forming a mechanical barrier against exogenous stimuli (Dayton et al. 1974, Brännström et al. 1979, Walton et al. 1989).

Tresiolan, a mixture of two siloxane esters, immiscible in water, polymerises to form an organo-siloxane resinous skin when applied to dentine. Several applications may be required to complete desensitization (Walton et al. 1989).

Dayton et al. (1974) evaluated several dental adhesives or enamel bonding agents (Nuva Seal, Enamelite, Restodent, Directon) and Zarosen, a varnish, in a twenty eight day clinical study involving 12 patients using thermal (a thermoelectric tooth stimulator) [Smith & Ash 1964 a,b]), mechanical (modified stimulating device [Smith & Ash 1964a,b]) and chemical (1M solution of sucrose) stimuli. All adhesive materials were reported to have reduced CDS with Nuva Seal and Enamelite producing significant reductions in response to thermal stimuli. Where the adhesive material had sheared off the tooth, the responses returned to their initial values.

Others have reported that the impregnation of dentinal tubules with

unfilled resins reduced CDS (Brännström et al.1979, Nordenvall & Brännström 1980, Nordenvall et al.1984). In 20 patients with follow up periods of two to twelve months, a drop of resin (Concise Enamel Bond) following acid etching of the dentine resulted in immediate and lasting reduction in sensitivity (Brännström et al.1979).

Following resin impregnation in the dog no neural activity was recorded (Närhi et al.1984). Copeland (1985) evaluated Scotchbond light cured dental adhesive/bonding agent and diluted Silux resin on 268 teeth using thermal (air stream) and mechanical (probe) methods during an eighteen month period. He reported 99% of teeth free of discomfort immediately following treatment performed under nitrous oxide analgesia, and 89% pain free at eighteen months.

Jensen and Doering (1987) evaluated Scotchbond and a preparation containing 0.42% NaF and 3.96% SCH in 38 patients over six months, using thermal (air spray) and mechanical (probe) methods. In most cases, Scotchbond desensitization provided immediate and lasting relief, whereas a single application of the NaF/SCH preparation burnished into dentine for three minutes was not as effective. It should be noted, however, that a single application may not necessarily reflect the true potential of the latter to reduce CDS over time.

Collins et al.(1990) in a single-blind clinical study involving 10 patients with 34 sensitive teeth evaluated an unspecified topically applied light cured resin over three weeks using thermal and mechanical methods (Orchardson and Collins 1987b), without sustained effect.

Yoshiyama et al.(1992) biopsied exposed root dentine in vital teeth to evaluate a new light curing resin (TMD-1) in 19 patients with 54 teeth. These investigators concluded that TMD-1 reduced sensitivity through tubule occlusion.

Felton et al.(1991) evaluated GLUMA (5% glutaraldehyde primer and 35% hydroxyethyl methacrylate) in 20 patients following preparation of teeth for full veneer crowns. Seventy six teeth were coated with either sterile water (control) or GLUMA in two applications of 30 seconds each. After 14 days response was evaluated by mechanical (Yeaple

probe), thermal (compressed air syringe) and chemical (saturated calcium chloride solution) stimuli. Significant reductions in sensitivity was observed for both GLUMA groups (intact and removed smear layer) compared to control, but no difference between the GLUMA groups.

Dentine adhesives have also been reported to reduce dentine permeability in vitro (Hansen et al.1991, Terkla et al.1991).

Javid et al.(1987) reported that cyanoacrylate application was more effective than 33% NaF in reducing CDS, when assessed by thermal (cold air) stimuli during a six week clinical study. This study, however, lacked suitable controls.

Positive, but variable results were also reported using Universal Bond and Scotchbond (Heymann et al.1987), Scotchbond 2 (Bastos et al.1991, Duke et al.1991a,b and Tenure (Taleghani & Leinfelder 1991) in class v erosion cavities.

Ianzano & Gwinnett (1992) reported that a single application of a hydrophilic dentine primer (N-phenyl glycine-glycine methacrylate and bis phenyl dimethacrylate) on 42 teeth in 7 patients was highly effective in reducing or eliminating dentine sensitivity, when assessed by cold and tactile (probe) methodology, over a six month period.

Reduction in sensitivity following placement of Glass Ionomer Cements (GIC) was observed by Low (1981) for periods up to fifteen months. Powell et al.(1990) reported that while glass ionomer restorations and restorations with composite resin and a dentine bonding agent significantly reduced sensitivity, they were also associated with increased sensitivity to air and cold respectively in 20-30% of the restorations at six months.

Recently a new GIC (GC Cervical cement, G-C International Corp Tokyo) has been marketed for CDS.

1.4.5.2. Varnishes

Various varnishes/liners, such as copalite have, been recommended for use in the treatment of CDS (Wycoff 1982).

Most cavity varnishes, however, appear to provide inadequate insulation against thermal conduction under restorative materials (Voth et al. 1966), while copal type varnishes (copal resin in an ether solution) are not compatible with resin based restorations and may interfere with the polymerization process (Tjan & Chan 1987, Tjan et al. 1987).

Recently a number of resin compatible cavity varnishes have been evaluated (Kaufman et al. 1982, Liberman et al. 1986, Tjan & Chan 1987, Tjan et al. 1987). UNIVAR/UNISEAL/MICROJOIN (Sci Pharm Duarte, CA USA) does not contain resin and consists of synthetic polyamino acids with a mixture of azeotropic solvents has been shown to occlude dentinal tubules (Kaufman et al. 1982, Liberman et al. 1986, Tjan & Chan 1987, Tjan et al. 1987).

A new tubule sealant (Barrier) has recently been advertised as a desensitizing agent; this material may, in fact, be the same material evaluated as a resin compatible varnish (UNISEAL).

Potassium oxalate has also been used as a cavity varnish/liner as well as a desensitizing agent in the treatment of CDS (Sandoval et al. 1989, Farmer & Cox 1990).

Fluoride varnishes such as Duraphat, an alcohol suspension of natural resins containing 5% NaF (2.26% F⁻), Fluor-Protector (a fluorsilane varnish) and Carex, containing 1.8% fluoride have been used in CDS (Clark 1982, Sutherland et al. 1989, Haugejorden & Nord 1991).

Clark et al. (1985) observed that a SCH/varnish (Duraphat) group demonstrated a 70% reduction in mean group pain score compared to the control group (sterile water), which demonstrated a 28% reduction. Fluoride varnish, together with SCH applied at home was more effective than SCH alone in treating CDS. There are several problems associated with this particular study, lack of objective methodology, small sample size, short duration and apparent discrepancy in the patterns of treatment. Both the control and SCH/varnish groups had bi-weekly treatments (up to eight treatments), whereas the SCH group was seen only at baseline and final examination, which may account in part for

the claimed efficacy of the SCH/varnish group over SCH alone. Also, differences between group scores at baseline meant that group changes were presented as percentage reductions from the initial scores. The investigators postulated that Duraphat's mechanism of action was by the formation of calcium fluoride and to some extent fluoroapatite, occluding dentinal tubules.

Positive results were also observed with Fluor-Protector (Collaert et al. 1991) and a sustained release device containing SCH in a hydrophobic polymer matrix (Mazor et al. 1989).

Caution should be applied to the interpretation of the findings on varnishes, particularly in the light of the small sample size, limited duration and lack of objective and reproducible methodology used.

Recently Tavares et al. (1992) reported on the effectiveness of a fluoride release resin (boron trifluoride BIS-GMA) in reducing CDS in 60 patients over a three month period. They reported that in groups with either the slow-release resin + unfilled resin or unfilled resin only, response to thermal and tactile sensitivity, compared to control teeth and baseline values, was greatly reduced or eliminated.

1.4.6. Miscellaneous treatment

Other treatment modalities include Burnishing/Instrumentation, Lasers, Restorative materials and Hypnosis.

1.4.6.1. Burnishing/Instrumentation

(See section 1.4.4.1.)

1.4.6.2. Lasers

Clinical lasers are of two types, soft lasers such as helium-neon (He-Ne), Gallium-arsenide (Ga-As) and gallium-aluminium-arsenide (Ga-Al-As) and hard lasers such as argon, carbon dioxide (CO₂) and neodymium yttrium aluminium garnet [Nd:YAG] (Frentzen & Koort 1990, Midda 1990, Midda & Renton-Harper 1991).

Frentzen and Koort (1990) conclude that for many clinical applications

the thermal side effects of hard laser lasing, such as charring and carbonation in both soft and hard tissues, particularly at high power, are limiting factors.

Izawa et al. (1991) investigated pulpal vascular reactions after Nd:YAG lasing and observed lasing caused a general decrease in pulpal vascular reaction and that the pulpal blood flow recovery was prolonged with increased laser duration. The dentine surface under SEM showed that power settings 30 and 50 mj/10 pulses/second at ten, twenty and thirty seconds produced crater-like impressions in the dentine surface. Fine holes or craters result from lasing depending on whether a focussed or defocussed laser beam is used (Paghdiwala 1991).

Slayton et al. (1992) reported that the effects of CO₂ laser irradiation on dentine permeability was comparable to the effects of oxalate application. SEM observation of the lased surfaces demonstrated a melted appearance of the created smear layer surface with no apparent damage to the underlying dentine.

Pashley et al. (1992b) reported that the effects of CO₂ lower and intermediate energy levels increased hydraulic conductance (Lp), possibly due to partial loss of the superficial smear layer and smear plugs and by crater formation making the dentine thinner. Higher energy levels, produced complete glazing of the crater surface and sealed the dentinal tubules beneath the crater, which decreased Lp, while at the same time removed a halo of 100µm of the smear layer around the crater, increasing Lp.

Crosa et al. (1991) concluded that structural changes in dentine induced by CO₂ lasing are less marked when application times are shorter.

Other investigators reported no adverse soft or hard tissue effects after lasing (Adrian et al. 1971, Adrian 1977).

Dederich et al. (1984) reported that the Nd:YAG laser was capable of creating root canal dentine fusion which would subsequently reduce dentine permeability.

Midda (1990) postulated that the laser energy sealed exposed dentine

tubules, possibly by creating a smear layer. White et al. (1990), however, investigated the effects of Nd:YAG lasing on hydraulic conductance (Lp) of dentine surfaces using the model devised by Goodis et al. (1989). Both pre- and post-lasing Lp values were determined following smear layer removal. Laser application did not alter the dentine surface (e.g., occlude tubules) sufficiently to cause changes in Lp in the studies of White et al. and Goodis et al., although recently White et al. (1992) reported that physical threshold modification of the dentine surface occurred at relatively low energy density levels with both Nd:YAG and Ho:YAG (Holmium) lasers. Goodis et al. (1992) also demonstrated that Nd:YAG was effective in reducing Lp using fetal calf serum to simulate dentinal fluid.

Several investigators evaluated the effects of lasing on CDS (Wilder-Smith 1988, Wakabayashi & Matsumotu 1988, Midda 1990, Midda & Renton-Harper 1991, Renton-Harper & Midda 1992).

Wilder-Smith (1988) used a VOCO PL 25 Helium-Neon laser for 2.5 minutes at 5HZ on three consecutive days in a one month clinical study involving 20 patients. 97 teeth which responded to cold air blast (dental air syringe) received soft laser therapy. The results highlighted the discrepancy between reduction in CDS as perceived by the patient and insignificant improvement demonstrated by a thermal stimulus. The soft laser treatment had little or no effect in relieving pain from CDS and the initial improvement, according to patients perception of pain, may have been due to a placebo effect.

The absence of a suitable control, together with the lack of accepted objective methodology as well as the relatively short duration of the study make evaluation difficult.

Wakabayashi and Matsumotu (1988) reported the short term effectiveness of a Ga-Al-As soft laser in reducing CDS. One hundred and thirty teeth were assessed by patient subjective response to a thermal stimulus (cold air blast). Details of methodology were unclear. If the patients were aware that active treatment was only on certain teeth, then this would have influenced their response and as such a placebo effect may

in part have influenced the reported effectiveness.

Renton-Harper and Midda (1992) evaluated a Nd:YAG laser (d Lase 300 American Dental Laser). An air jet stimulator (Hypersensitivity tester machine) based on the type used by Orchardson and Collins (1987b) was used to evaluate response to cold air. The initial air flow tolerable to patients in the lased and non-lased groups was 1.6 and 1.9 seconds respectively. Following lasing, the air flow time average was 3.7 seconds. Subsequent evaluation at 3, 7, and 14 days indicated an overall reduction in sensitivity, in that tolerance to the thermal stimulus increased, compared to the air flow times of the control group, in which there was little change in sensitivity throughout the study.

Although the study indicated that the Nd:YAG laser used at power levels up to one watt was an effective and reproducible tool in the treatment of CDS, the methodology employed in the study may be criticised. The use of a constant air stream has been shown to dessicate the tooth surface and may also involve other effects apart from a purely thermal stimulus (**section 1.3.3.**). Subjective evaluation of CDS has also been a problem. As patients were also aware which teeth had been lased, it is reasonable to suggest that a placebo effect may have influenced the results. No assessment of the long term effectiveness of the Nd:YAG laser in reducing CDS was attempted by the investigators.

To date, few studies have attempted to evaluate the effectiveness of soft and hard lasers in reducing CDS and while efficacy of the laser has been claimed, a strong placebo effect cannot be ruled out.

1.4.6.3. Restorative materials

(See section 1.4.5.1.)

1.4.6.4. Hypnosis

Recently hypnosis has been suggested by Starr et al. (1989) for patients with CDS. They conducted a four week study involving 8

patients in which all patients received three to five minutes formal hypnotic induction using progressive relaxation until analgesia or altered sensation of one hand was obtained. Through hypnotic suggestion, this altered sensation was transferred from the hand to the appropriate side of the mouth. It was reported that all patients demonstrated significant improvement in symptoms over the four week period. 5/8 patients, however, reported the use of analgesics during the study, although this was not correlated to pain relieving effect according to the patient questionnaire. These investigators claimed hypnosis may be effective in reducing CDS, although they recognised that further studies were required.

This study, however, did not utilise accepted methodology for the assessment of CDS and may also be criticised for having relied solely on subjective response through questionnaire evaluation.

1.4.7. Summary

To date no single desensitizing agent or therapeutic technique, despite claims to the contrary, has fully satisfied Grossman's original criteria (1935).

One of the difficulties in evaluating the claims made concerning the supposed efficacy of the various desensitizing dentifrices is that many of the earlier studies were based on testimonial rather than scientific evidence. Indeed it is very difficult to determine clinically whether a desensitizing agent has been successful in reducing CDS on the basis of its mode of action alone. There is also a problem with classifying these agents on the basis of their supposed mode of action as proposed by Ong (1986) and Scherman & Jacobsen (1992). For example, SCH is claimed to act both as a protein precipitant and tubule occluding agent. Evidence for SCH's mode of action, however, is lacking. This statement may also be extended to cover most, if not all, of the agents reviewed in this thesis. An alternative classification based on whether the agent's mode of action is through tubule occlusion or alteration of sensory nerve activity through raised K^+ concentration (direct ionic

diffusion) would appear to provide a better classification system, but again evidence is still lacking as to whether these agents desensitize the tooth (in vivo) in the manner claimed by investigators.

Various in vitro studies have highlighted discrepancies between laboratory findings and the claims of clinical efficacy of desensitizing dentifrices. Some in vitro studies have suggested that the abrasive components of the dentifrice may be responsible for the desensitization of dentine through tubule occlusion rather than the active ingredient per se. Several laboratory studies have also failed to substantiate the claims of various desensitizing agents, such as KNO₃ and SCH, although it is possible that other mechanisms of action may be responsible for their reported clinical success.

The use of the in vitro dentine disc model as a screening/testing procedure for potential tubule occluding agents would appear valid, a suitable alternative to the animal model for screening/testing potential agents which may alter nerve activity does not appear to be forthcoming.

Recent in vitro studies have also highlighted a number of potential agents, such as the oxalates, although these claims too need to be substantiated in clinical trials against existing recognised agents. Other alternative forms of treatment have been proposed, although little evidence is forthcoming for methods such as hypnosis. Laser technology has been reported as being successful in relieving CDS, although further research is needed using more objective methodology, as recommended by the American Dental Association (1986). In cases where persistent, long term sensitivity has been a problem, restorative materials such as glass ionomer cements and adhesives have been reported to reduce CDS. Correct diagnosis and elimination of aetiological factors associated with CDS may also prevent the occurrence/recurrence of CDS e.g., dietary counselling and non-traumatic brushing techniques.

Because of the subjective nature of this complaint, objective evaluation of known or potential desensitizing agents has proved

difficult. Recent improvements in methodology may help to overcome such difficulties, although there is still a need for further development in this field of clinical research which, together with well-controlled long term clinical studies will enable more accurate and objective assessment of the efficacy of potential desensitizing agents used in the treatment of CDS.

On the basis of reviewing the available literature, however, it is apparent that there are still disagreements over what is the best methodology to use to evaluate patient response and whether there are true positive or negative controls available which could act as the gold standard when testing new desensitizing dentifrices. Until such deficiencies are addressed the problems of evaluating the efficacy of these desensitizing agents in reducing CDS will persist.

CHAPTER 2

Clinical evaluation of two SCH dentifrices

Introduction

Efficacy of desensitizing dentifrices has been previously attributed to the active ingredient, although several investigators have indicated that the abrasive component may be responsible for this effect (**section 1.4.2.**). The effectiveness of various dentifrices such as SCH in reducing CDS has also been questioned (**section 1.4.2.**). Several investigators (Addy *et al.* 1987b, Jackson *et al.* 1989, 1990) have reported that silica-based products containing SrAc_2F were more effective in controlling CDS than a dentifrice containing SCH and the abrasive diatomaceous earth. The results of these studies would, therefore, appear to suggest that the inclusion of a silica-based abrasive system into a desensitizing dentifrice (irrespective of the so-called active ingredient) would be beneficial in terms of reducing CDS.

The purpose of this clinical study, therefore was to determine whether changing the abrasive system of a desensitizing dentifrice (SCH) would have any effect on the efficacy of the two SCH dentifrices (silica-based/diatomaceous earth) in reducing CDS.

2.1. Calibration Studies

Equipment Evaluation and Reproducibility Studies

Introduction

Prior to the commencement of the main clinical study a number of in vitro and in vivo studies were performed in order to evaluate the equipment to be used as well as to ensure reproducibility of the variables to be assessed.

2.1.1. Tactile stimulus (Yeaple probe)

An electronic pressure-controlled probe device, the Yeaple probe, has

been developed to enable controlled force probing (**Fig. 2.1**). The probe utilises a readily variable electro-magnetic force transducer to control the pressure applied at the tip of a removable probe. The device was primarily designed to function as a pressure controlled periodontal probe (Polson et al. 1980). For the purpose of the clinical studies, the standard tip used was a pre-sterilised Williams 14W periodontal probe tip with a rounded end 0.30-0.35mm in diameter (**see probe tip section**). The force applied at the tip is altered by varying the current applied. When a pre-set probing force is reached a red light is illuminated on the control panel and sound emitted, and an electromagnet releases the probe tip to control the pressure applied. The probe handle is 1.27cm in diameter and 15.5cm in length (attached probe tip and housing 2.54cm) and is attached to the control panel by a flexible cord (**Fig. 2.2.**). The cervical area of the sensitive tooth to be tested was isolated and the probe tip stroked across it. The initial setting was 10 gram weight which was increased by 5 gram weight increments until the patient indicated discomfort or until a maximum setting of 70 gram weight was reached. Change at later visits during the clinical study was evidenced by altered response to a given probe setting.

Probe tip Evaluation

The original Yeaple probe tip was a No. 19 U.K. explorer. Following a laboratory investigation using extracted teeth it was observed that this probe tip caused superficial surface damage to the root surface particularly at the higher probe settings greater than 40 gram weight. In order to ascertain whether such damage could be minimized, a further investigation was carried out substituting the original explorer tip with a Williams 14W periodontal probe tip with a rounded end 0.30-0.35mm in diameter.

Procedure

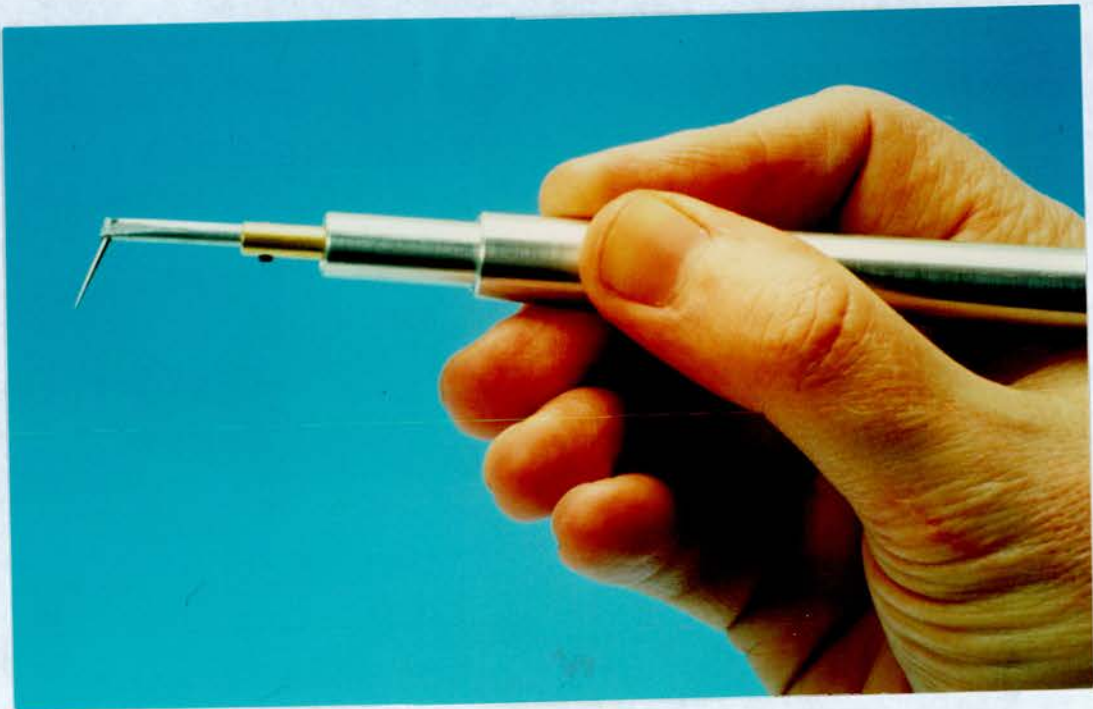
The root of an extracted tooth was tested for surface damage following

Figure 2.1.



Yeaple probe

Figure 2.2.



Yeaple probe handle showing tip

the application of a sharp No. 19 explorer tip and a modified tip with a rounded end 0.30-0.35 mm in diameter. Using a range of pre-set force settings (10, 20, 30, 40 and 50 gram weight), the probe tip was moved perpendicular to the root surface. At each stage the tooth was examined for evidence of surface damage, and if apparent the tooth was stained with a food dye and photographed (**Fig. 2.3.**). This procedure was then repeated using the modified tip. Due to light interference on the tooth surface making the scratch marks difficult to identify for staining, the procedure was modified to stain the tooth first. The sharp tip appeared to produce scratches discernible to the eye only at forces > 40 gram weight (**Fig. 2.4.**), whereas no discernible surface damage was observed with the modified tip (**Fig. 2.5.**).

On the basis of this simple in vitro study, the protocol for the clinical study was amended to include the use of the modified tip.

Yeaple probe Calibration

The manufacturer of the Yeaple probe (Vine Valley Research, Middlesex, N.Y., U.S.A.) stated that probe settings could be controlled to within ± 1 gram weight. The investigator calibrated the probe (Model 200A Serial No 1106) (**Fig. 2.1.**) prior to the commencement of the clinical study. The handpiece of the probe was clamped in a ring stand with the tip held perpendicular to the Sartorius 1002 MP top-loading digital balance (Brinkman Instruments Co., Div. of Sybron, Westbury N.Y., U.S.A.) (**Fig. 2.6.**) placed on a small platform which was slowly raised to contact the probe tip. The procedure was repeated 5 times for each mA reading (19, 35, 45, 53, 60, 66, 72, 77, 82, and 87) and the corresponding reading in gram weight recorded. The results of the pre-study probe calibration was recorded and a calibration curve charted (**section 2.3.1.**).

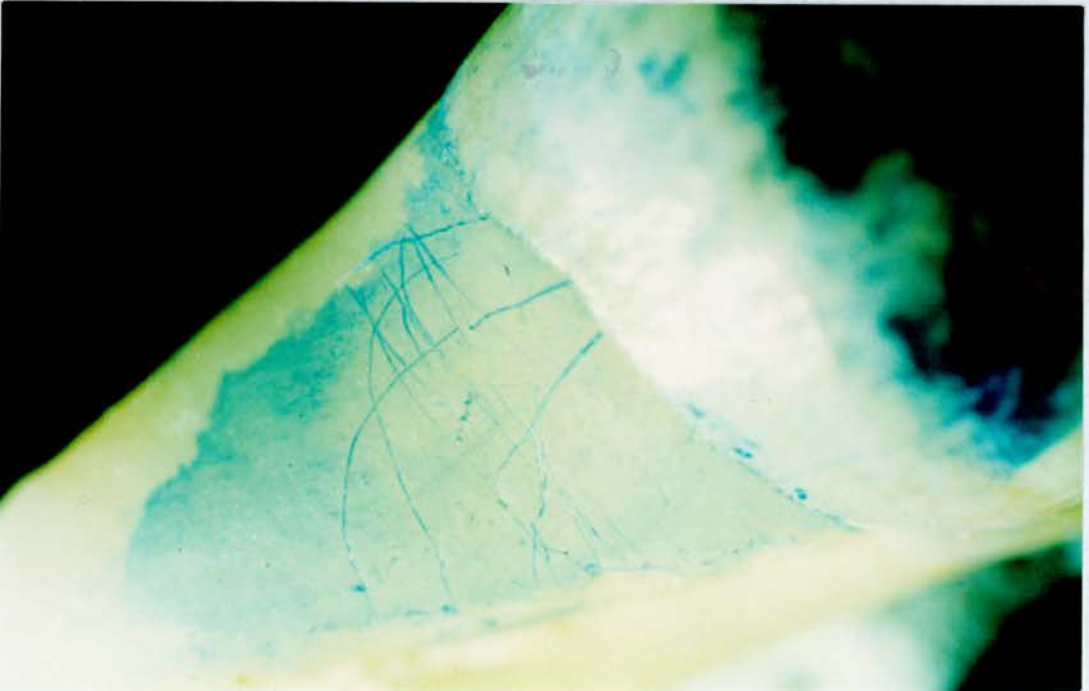
The Yeaple probe was similarly calibrated using 4 selected mA readings (19, 45, 60, 72) prior to each clinical session (**section 2.3.2.**).

Figure 2.3.



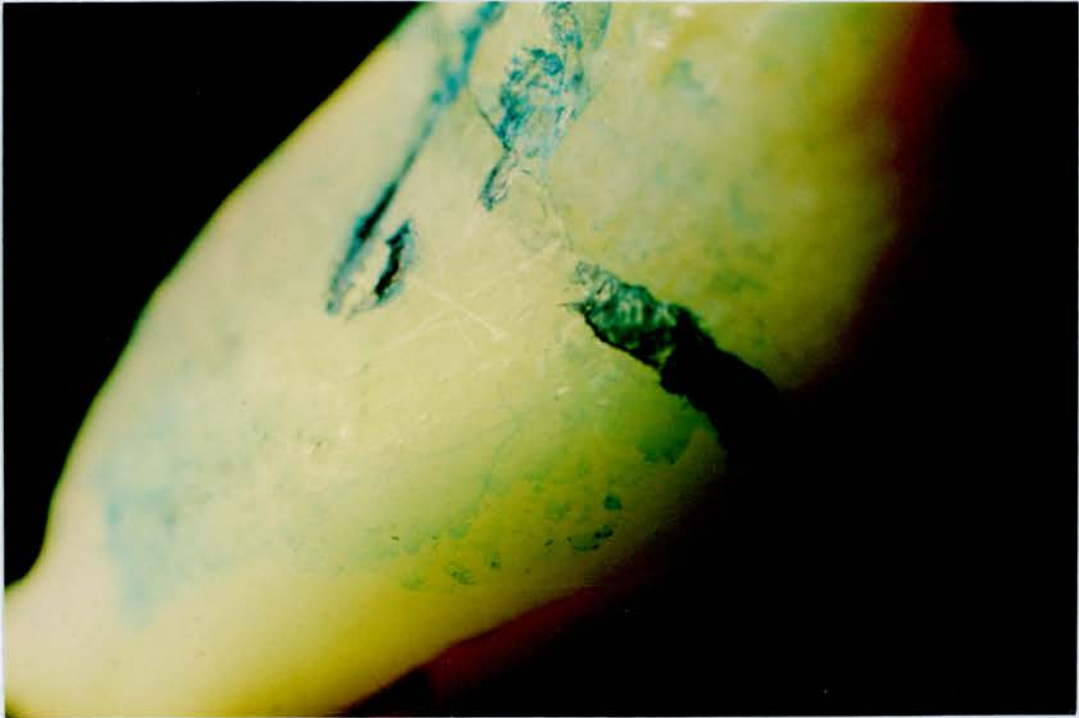
Unstained tooth showing surface scratches following probing with a sharp probe tip (No. 19 U.K. explorer)

Figure 2.4.



Stained tooth showing surface scratches following probing with a sharp probe tip (No. 19 U.K. explorer)

Figure 2.5.



Stained tooth showing no discernible surface damage following probing with a modified tip (Williams 14W periodontal probe)

Figure 2.6.



Yeaple probe and Sartorius balance

2.1.2. Thermal Stimulus (cold air)

The response to thermal stimuli was assessed using a one second application of cold air delivered from a standard dental unit syringe at 40-65 p.s.i. at a temperature of about 19°C. Ong & Strahan (1989) previously calibrated air temperature by blowing the air into a custom made Vel-mix casting containing a clinical thermometer. Air temperature was $24.5^{\circ}\text{C} \pm 1.5^{\circ}\text{C}$. Current studies using a thermocouple (**Chapter 6**) would indicate a temperature range of 19°C-24°C ($\bar{x} = 23.2^{\circ}\text{C}$). Prior to testing each tooth was isolated from adjacent teeth mesially and distally by the investigator through use of fingers and/or cotton wool rolls. The air was directed perpendicular to the exposed root surface of each test tooth, the syringe tip being 1cm away from the tooth.

2.1.3. Patient Subjective Response

Patient subjective response was recorded using Visual Analogue Scales (VAS cm) and utilised for Overall sensitivity, tactile and air subjective responses. The investigator ensured that the patient was familiar with the VAS procedure for each of these three responses prior to testing. The pain intensity from the test stimuli was indicated by the patient placing a mark on a line 10cm in length (**Fig. 2.7.**). The distance from the 'no pain' end provided an estimate of pain as perceived by the patient and constituted a sensitivity score which was then recorded on the relevant clinical form.

Prior to the commencement of the main clinical study, a small calibration study involving 5 patients (1M, 4F, mean age 40.7, 95% C.I.: 38.86 - 46.48 years) evaluated CDS on two occasions (0 and 7 days) using the three methods of assessment outlined in the main study protocol (**section 2.3.1.**).

2.2. Materials and Methods

Forty-nine patients were originally enrolled into the main study (**Fig.2.8.**). Nine were subsequently excluded. One failed to disclose a

Figure 2.7.

OVERALL SENSITIVITY SCALE

Visit (circle one) B2, Week 2, Week 4, Week 8

To the subject: You will be given this form to complete prior to the examination. You are asked to rate the severity of the tooth pain that you experience during your everyday routine from hot/cold food or drink, air, tooth brushing, sweet and sour food. Now place a mark through the line below to indicate the severity of tooth pain you have experienced.

No Pain _____ Unbearable Pain

Visual Analogue Scale (VAS) form
(Used for Overall Sensitivity, Tactile & Air Sensitivity evaluation)

medical problem, one had periodontitis, one did not respond to the test stimuli, one failed to return following screening and five were unable to attend for all visits. Forty subjects completed the 8-week clinical study. The investigation was a double-blind, 2 way comparative parallel study of 40 patients mean age 42.8 years (**Table 2.1., Fig. 2.9.**). Subjects were randomly assigned to one of the two treatment groups using a computer-generated randomisation code.

2.2.1. Inclusion Criteria

Selection of subjects was restricted to individuals who presented with CDS, accompanied by cervical erosion, abrasion and/or gingival recession on at least one tooth for tactile stimulus and two for cold air stimulus on suitable teeth anterior to the second molar. Sensitive teeth without restorations were preferred, although teeth with restorations were included provided the restorations were no greater than one half of the distance through dentine in anterior, premolar and first molar teeth. Any restoration margins were at least 5mm from the area of sensitivity. Decision to include such teeth was made on the basis of clinical as well as radiographical (OPT) evaluation. Subjects were included who, during the baseline examination, experienced sensitivity to a tactile probe setting of 10-50 gram weight and recorded a Visual Analogue Scale score of 3-8cm following application of a cold air stimulus. All showed at least one tooth sensitive to the Yeaple probe and two to the dental air syringe, although not necessarily the same teeth.

2.2.2. Exclusion Criteria

Subjects with chronic systemic disease or a history of gingival surgery within the previous six months were excluded, as were patients who were pregnant or lactating, or who were on any medication. Teeth with suspected pulpitis, caries or cracked enamel were excluded, as were all teeth with defective restorations and those used as abutments. Subjects with Gingival Index (Löe 1967) > 1 for the gingivae of

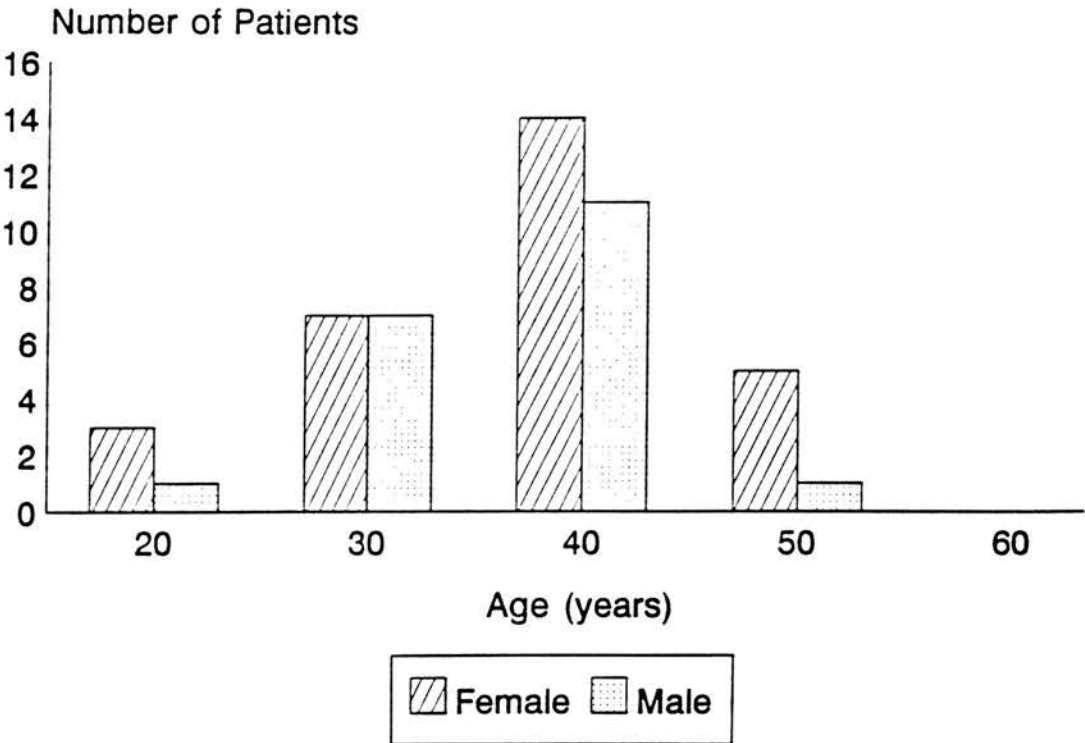
Table 2.1.Patient Data for 8 and 20-week clinical studies

Sex	N	Test Gp Mean Age (SD)	N	Control Gp Mean Age (SD)	N	Total Mean Age (SD)
Female	13	42.6 (11.38)	12	43.9 (8.24)	25	43.2 (9.81)
Male	7	40.3 (4.88)	8	43.8 (3.84)	15	42.1 (4.57)
Mean	20	41.8 (9.52)	20	43.8 (6.69)	40	42.8 (8.18)

Test Gp = Silica-based

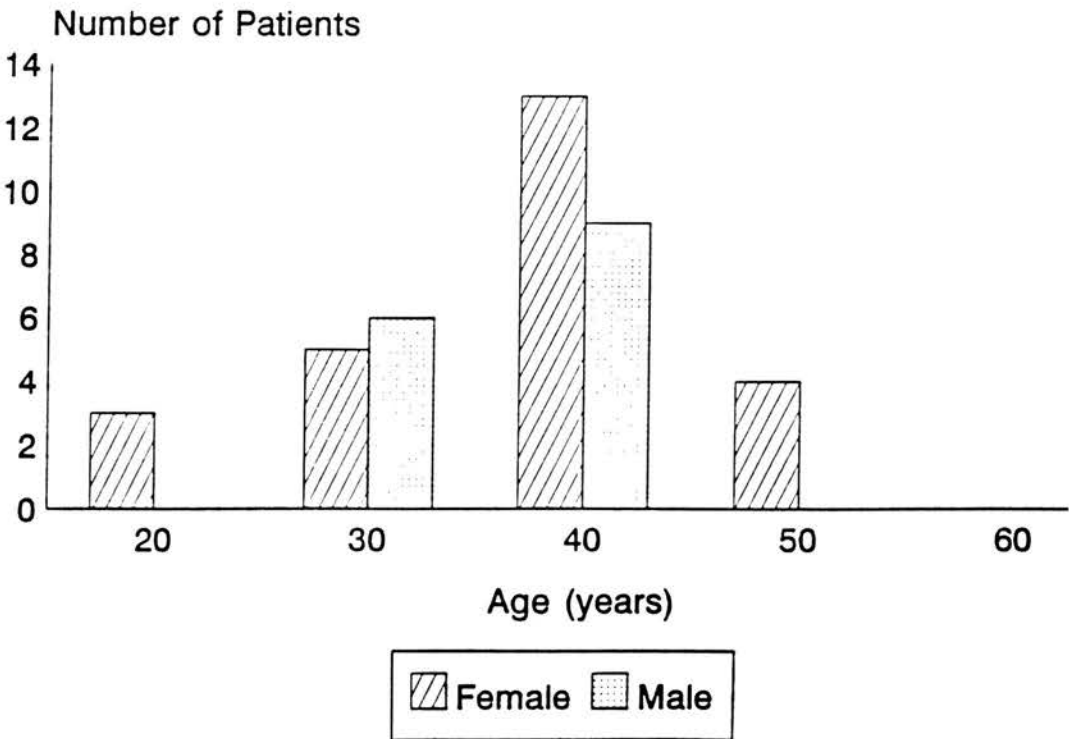
Control Gp = Diatomaceous earth

Figure 2.8.



Distribution of sensitivity by Age & Sex (49 patients)

Figure 2.9.



Distribution of sensitivity by Age & Sex (40 patients)

prospective sensitive and adjacent teeth, or > 2 for non-study teeth were also excluded. Subjects using a desensitizing dentifrice agreed to refrain from using same for at least two months prior to the study. Fourteen subjects (7 male, 7 female) were in this category and a fluoride dentifrice was substituted for the desensitizing paste. The subjects were instructed to continue their normal daily oral hygiene practices.

2.2.3. Screening

Following approval of the Institute and Hospital Joint Research and Ethics Committee and individual voluntary written informed consent, subjects completed a questionnaire concerning their sensitivity complaint (**Fig. 2.10.**). Subjects thus screened were examined for baseline sensitivity using both tactile and cold air stimuli. Sensitive teeth were initially detected with a No. 6 straight probe cervically on each tooth anterior to the second molar. Ten minutes later the investigator assessed the tooth response to cold air using the standard dental air syringe at 40-65 p.s.i. at a temperature of 19-24°C.

2.2.4. Procedure for measuring CDS

Tactile Method (Yeaple probe - Modified)

The Yeaple probe (Vine Valley Research, Middlesex, N.Y., U.S.A.) was modified to accept a tip with rounded end 0.30mm-0.35mm diameter (Williams 14W) (**Fig. 2.11.**). The probe is designed to deliver a pre-set force when the tip is applied perpendicular to the cervical labial surface (Polson et al.1980). The initial probe setting was 10 gram weight and the settings were adjusted in 5 gram weight increments continuing up to the point at which discomfort was just felt and the probe setting noted. The maximum probe setting was 70 gram weight. If following the two baseline measurements the subject did not perceive any discomfort at that force, a score of 70 was recorded. The subject

Figure 2.10.

HISTORY

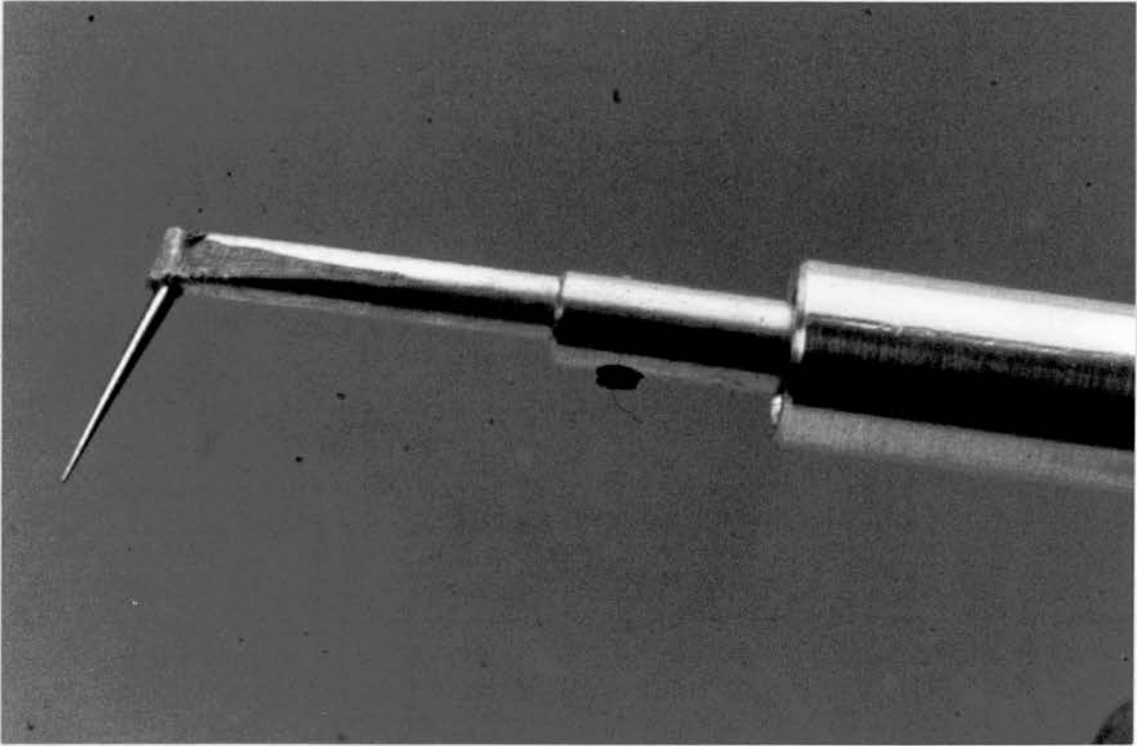
1. Date of birth

Day Month Year
2. Sex (tick one box) Male ☐ Female ☐
3. To what are your teeth sensitive? _____ (eg not, cold, sweet, sour, touch, nothing).
4. Your normal oral hygiene
 - a) Which toothpaste do you normally use? _____
 - b) How many times a day do you brush? _____
 - c) Other oral hygiene aids used? _____
(eg. floss, mouthwash)
5. Which hand do you use to brush your teeth? _____
6. Does your toothbrush have nylon or natural bristles? _____
7. Do you often eat or drink acid foods? (Yes or No) _____
If yes, state which (eg. orange, grapefruit, tomato juice etc.) _____
8. Have you had periodontal surgery within the last 6 months? _____
9. Have you had a whole mouth X-ray taken recently, and if so, by whom? _____
10. Do you avoid brushing certain areas because of pain in the area? _____
11. Have you had professionally administered treatment for sensitivity? (Yes or No) _____
If yes: What kind (resin, fluoride) _____
Which tooth _____
Date applied _____
12. Describe your tooth hypersensitivity in your own words _____

13. Circle the words on the next sheet which best describe your pain.

Sensitivity Questionnaire

Figure 2.11.



Modified Yeaple probe tip (Williams 14W periodontal probe tip)

was also asked to rate the perception of sensitivity experienced during application of tactile probe by placing a mark on a 10 cm line on a Tactile Sensitivity Scale form (**Fig. 2.7.**). The distance of the mark from the 'no pain' end provided an estimate of pain perceived by the subject and constituted a Tactile VAS score. The Yeaple probe was calibrated prior to each clinical session using a Sartorius 1002 MP top loading digital balance (Brinkman Instruments Co., Div. of Sybron, Westbury, N.Y. U.S.A.) to obtain a correlation of the probe meter readings in DC microamperes and the grams of force (**section 2.1.1.**).

Cold Air (Thermal Method)

After a ten minute interval the test tooth was isolated and patient response was assessed following a one second application of cold air (dental unit syringe 19-24°C, 40-65 p.s.i.) directed perpendicular to the exposed root surface. Using the principle of Visual Analogue Scale Scores (0-10) air pain intensity was indicated by the subject placing a mark on a 10cm line on a Subject Air Sensitivity Score Form (**Fig. 2.7.**). The distance of the mark from the 'No pain' end provided an estimate of pain perceived by the subject and constituted an air sensitivity score.

Subjective Reporting of Pain-Baseline

Subjects were asked to rate their perception of sensitivity to hot/cold food and drink, air, toothbrushing and sweet and sour food by placing a mark on a 10cm line (**Fig. 2.7.**). The distance of the mark from the 'No pain' end provided an estimate of the overall severity of pain perceived by the subject. After approximately one week, a second baseline determination was made repeating the above procedure.

2.2.5. Test Product Assignment

Assignment of subjects to experimental cells was by a computer-generated random number code. Each individual coded kit contained two toothbrushes (Sensodyne Search 4) and tubes (3 x 45ml) of the test

dentifrices. Dentifrices were closely matched with respect to taste, colour, consistency and appearance and dispensed double-blind. Subjects were directed to brush twice each day, morning and evening, in their usual manner, with the brush supplied, for 56 consecutive days, using only the assigned dentifrice.

Each patient was instructed to place an inch length of toothpaste on the wet toothbrush and to brush all surfaces of all teeth for at least one minute. Each subject recorded his/her daily brushing in a diary which was provided (**Fig. 2.12.**). All assigned products were weighed before and after use by the investigator to assist in determining compliance.

The diaries were checked at each visit by a third party who also distributed the assigned products. All patients attended all appointments, and on or close to day 56 with residual toothpaste. Recorded non-compliance with regard to dentifrice use was rare.

2.2.6. Data Analysis

All data were tested for Normality using a normal scores transformation and plotting the result against the original data. A normal distribution was indicated by a reasonably straight line plot with no marked concavity or convexity. All data proved to be normally distributed with exception of tactile force which was skewed to the right. A logarithmic transformation was, therefore, used to normalise these data and stabilise the variance. For this variable, therefore, descriptive statistics only were provided for the raw (untransformed) data and analysis was carried out on the log-transformed data. Normality tests also detected a marked 'outlier' reading in the test group 'Baseline minus 2-week' data. Analyses were, therefore, performed both excluding and including the 'outlier'. In the event, inclusion did not affect the overall trend of the data, but data excluding the 'outlier' were taken as being more reliable.

Subject-based (n = number of subjects)

Paired t-tests were utilised for each treatment cell to determine if differences between readings at baseline and at scheduled examination times were statistically significant at the 95% confidence level. Similarly, at each time point any differences between the dentifrices and their effects on sensitivity scores were tested for statistical significance by means of a two-sample t-test. Confidence intervals were also calculated and only probabilities of less than or equal to 0.05 were considered to indicate a significant difference between means.

2.2.3. Results

2.3.1. Calibration Study

Yeaple probe calibration values (Laboratory)

A comparison of the manufacturer's and the investigator's calibration is provided (Table 2.2.a-c., Fig. 2.13.).

Results from the pre-clinical calibration study (Table 2.3.) indicated that there were no significant differences between score values for Overall Sensitivity or Cold Air stimulation when treated as paired and unpaired samples.

For Overall Sensitivity (paired $t = 1.67$, 8 df, unpaired $t = 1.20$, 4 df), and Cold Air (paired $t = 0.26$, 8 df, unpaired $t = 0.52$, 4 df) respectively. It was not possible to demonstrate correlation between the two visits; for Overall Sensitivity $r = 0.6748$, $r^2 = 0.4554$, that is 45% of variation of scores from the second visit, can be explained by the variation of the first. For Cold Air $r = 0.8378$, $r^2 = 0.7019$, that is 70% of the variation of scores from the second visit can be explained by the variation of scores from the first. For Yeaple probe score values, however, there was insufficient data (non-parametric) to analyse the result and even when log transformed there was no significant difference ($t = 2.305$, 4 df). No correlation of scores from the two visits was attempted as the insufficient data values were

Table 2.2.(a)Manufacturer Yeaple probe calibration values (1988)

mA	19	35	45	53	60	66	72	77	82	87
gm*	10	20	30	40	50	60	70	80	90	100

* gram weight

Table 2.2.(b)Summary: Investigator calibration readings (1989)

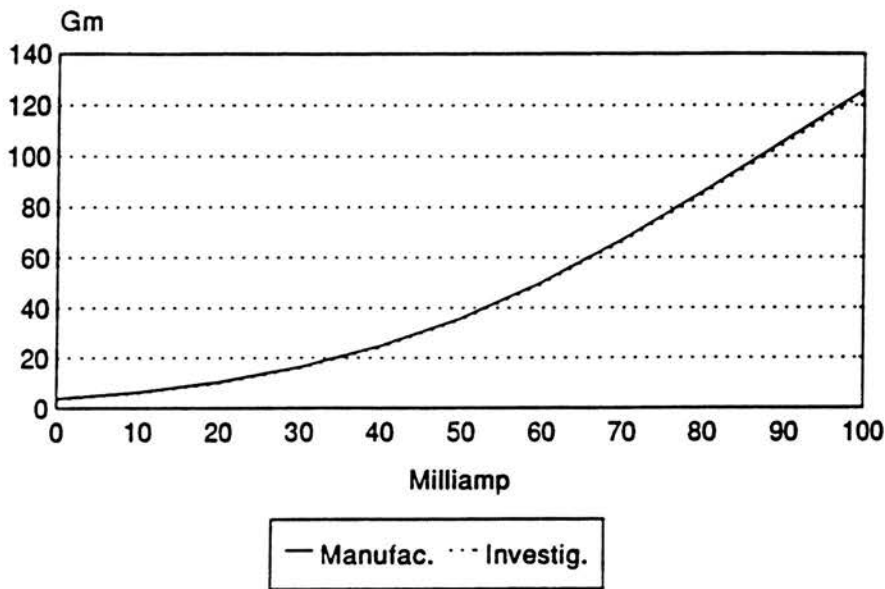
mA	19	35	45	53	60	66	72	77	82	87
gm*	9.7	19.7	29.3	39.6	49.8	59.3	68.9	79.1	88.4	99.7

*gram weight

Table 2.2.(c)Investigator calibration values (gram weight) from set mA readings

10	20	30	40	50	60	70	80	90	100
10	20.2	29.3	38.2	49.9	60.4	69.0	81.9	90.2	98.6
9.6	19.5	29.1	38.6	50.0	59.2	68.9	78.5	87.6	100.7
9.8	19.4	29.3	40.0	49.0	60.6	68.2	78.7	88.0	99.1
9.5	19.9	29.6	38.9	50.3	58.3	68.8	78.1	87.5	100.0
9.6	19.7	29.5	38.4	49.8	58.2	69.0	78.5	87.2	100.1
9.7	19.7	29.3	39.6	49.8	59.3	68.9	79.1	88.4	99.7

Figure 2.13.



Comparison of Manufacturer & Investigator calibration values (gram weight) for the Yeaple probe

Table 2.3.

Pre-clinical calibration study (5 patients)

Overall Sensitivity (cm)		Cold Air (cm)		Yeaple probe (gm*)	
Visit 1	Visit 2	Visit 1	Visit 2	Visit 1	Visit 2
6.1	4.3	8.5 4.4	8.1 1.8	10	10
5.1	1.4	4.3 2.4	7.0 3.8	15	15
7.6	8.2	7.7 8.9	7.3 8.3	15	10
3.7	2.3	1.6 1.3	1.9 0.7	30	15
3.5	4.6	2.7 2.5	2.0 7.0	30	15

*gram weight

considered unreliable for further treatment.

2.3.2. Clinical Study

Distribution of teeth responding to Probe and Cold air blast

Forty nine patients were screened for inclusion in the clinical trial (**Fig. 2.8.**). Two hundred and twenty three out of nine hundred and eighty four teeth responded to probing. The proportion of teeth responding to this stimulus is shown in **Fig. 2.14.**. Two hundred and eighty three out of nine hundred and eighty four teeth responded to cold air blast. The proportion of teeth responding to this stimulus is shown in **Fig. 2.15.**. Forty patients who agreed to participate in the clinical study responded to probe and cold air stimuli as follows: 188/893 teeth responded to probing. The proportion of teeth responding to this stimulus is shown in **Fig. 2.16.**, 234/893 teeth responded to cold air blast. The proportion of teeth responding to this stimulus is shown in **Fig. 2.17.**.

Distribution of Test teeth responding to Probe and Cold air blast

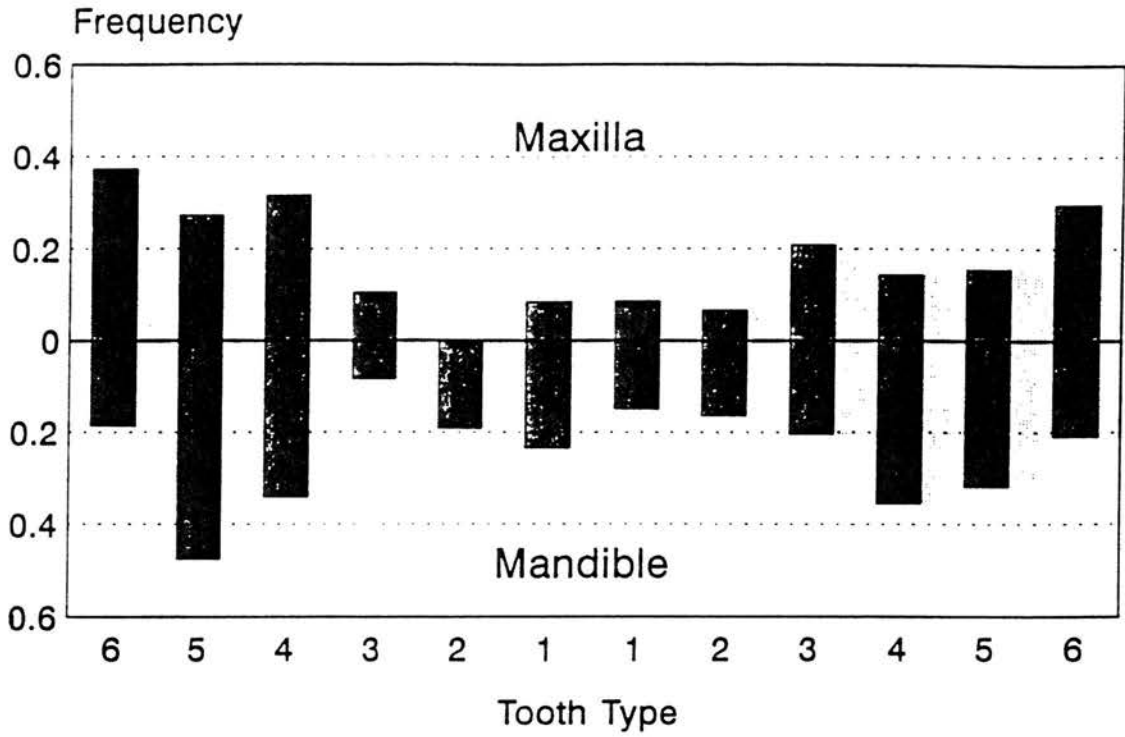
Seventy seven teeth responded to probing (Yeaple probe) and 80 teeth responded to cold air blast. The proportion of teeth responding to these stimuli are shown in **Figs. 2.18.-2.19.**.

Patient reported frequency to various pain stimuli

Patient-reported response to various pain stimuli is shown in **Figs. 2.20.a-b.** 34/40 (85%) and 40/49 (81.6%) patients also stated that they regularly took acid food/drinks.

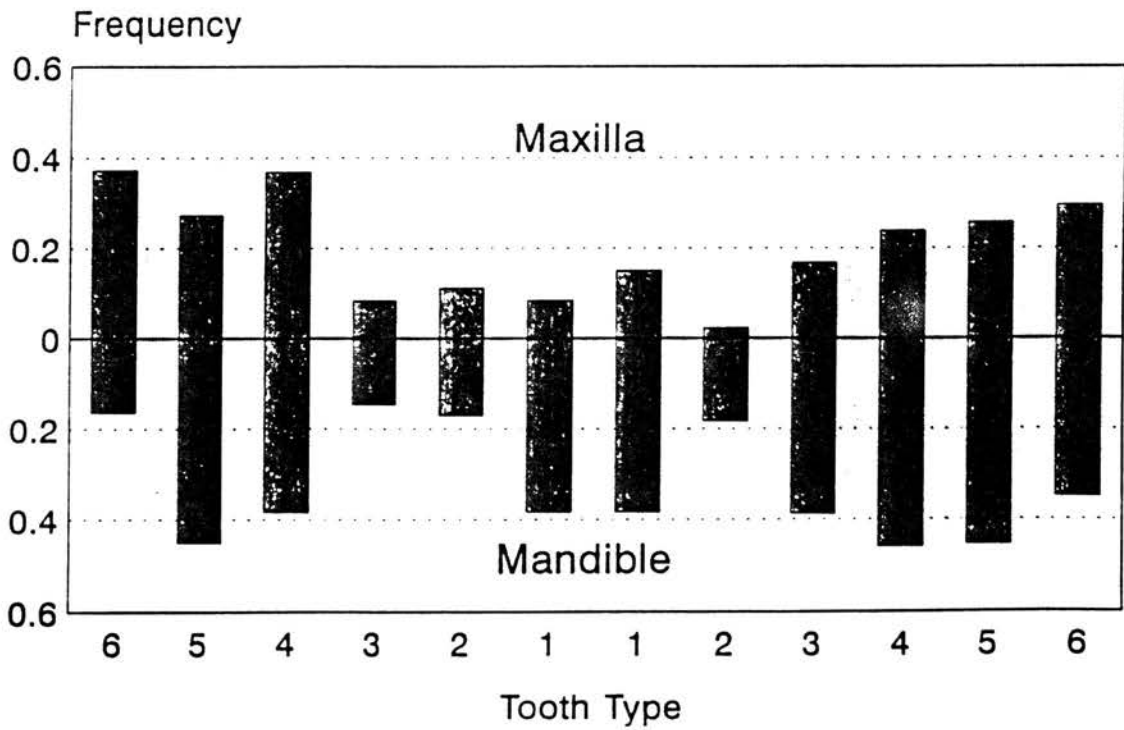
Forty subjects (15 male and 25 female, mean age 42.8 (SD 8.2) years, completed this study (**Table 2.1.**). No changes were observed in the oral tissues of any subject in either group over the 8-week study period, nor were side-effects or untoward reactions reported to or observed by the investigator.

Figure 2.14.



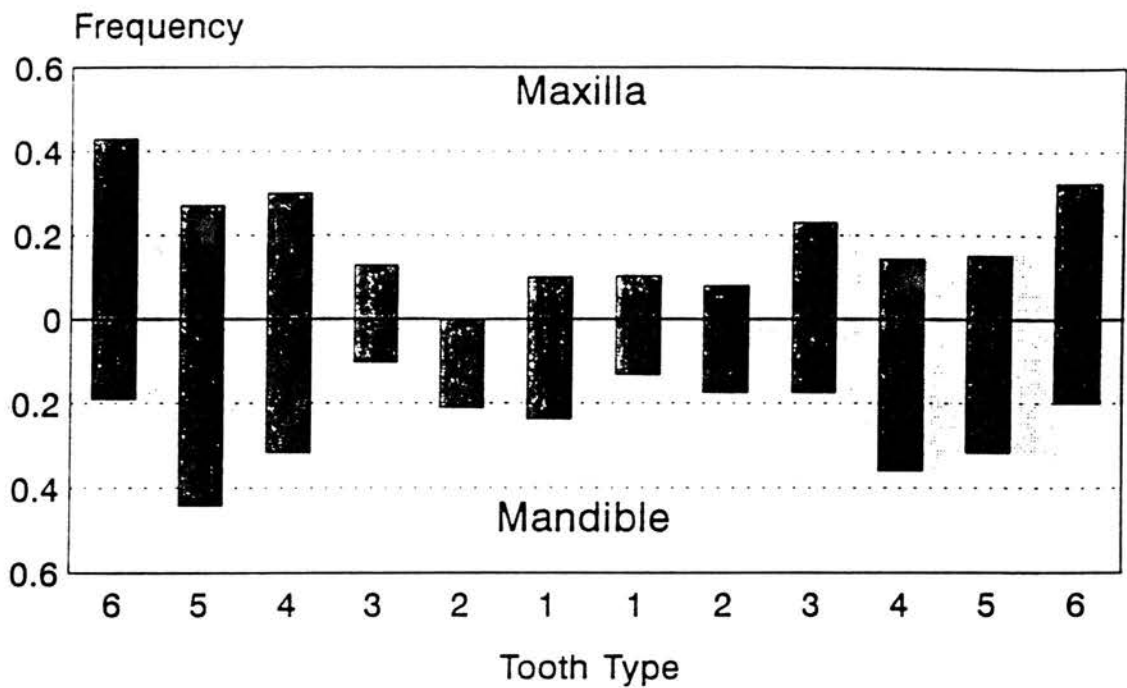
Proportion of teeth showing response to tactile stimulus (explorer probe) Buccal surfaces (49 patients)

Figure 2.15.



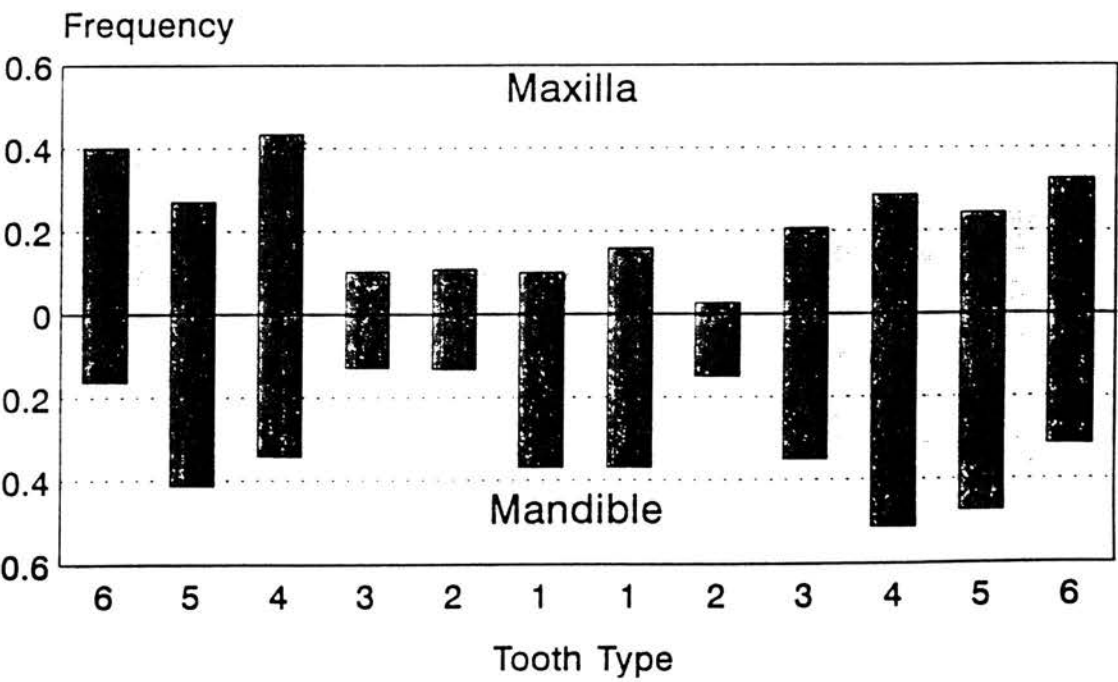
Proportion of teeth responding to thermal stimulus (cold air blast) Buccal surfaces (49 patients)

Figure 2.16.



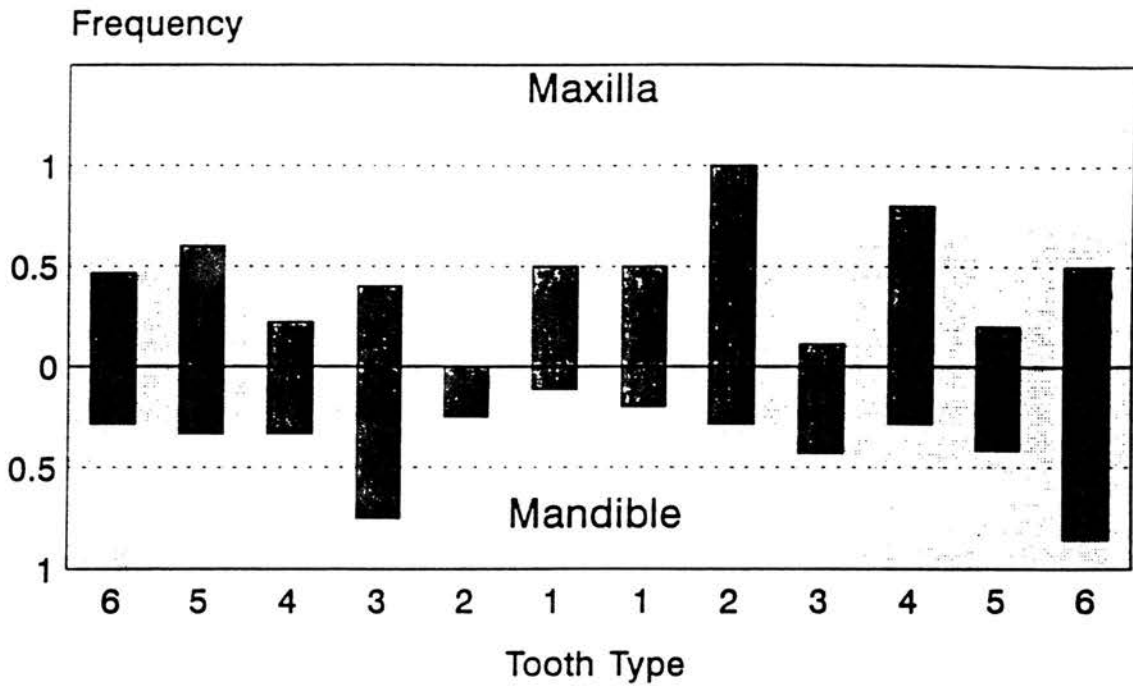
Proportion of teeth responding to tactile stimulus (explorer probe)
Buccal surfaces (40 patients)

Figure 2.17.



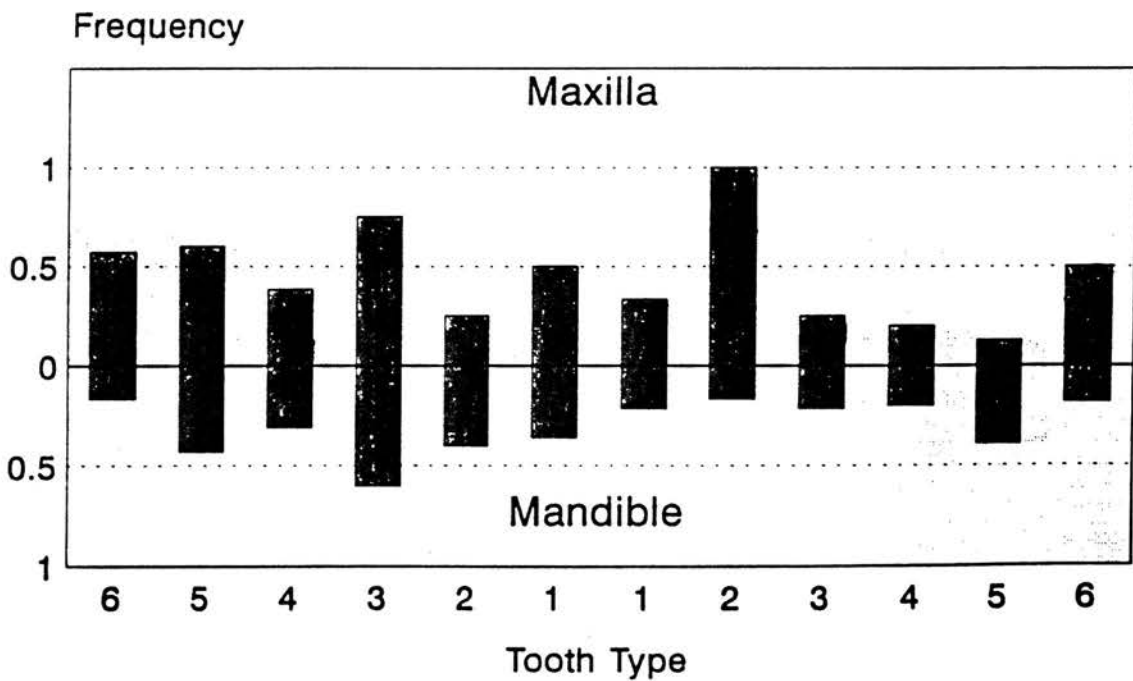
Proportion of teeth responding to thermal stimulus (cold air blast)
Buccal surfaces (40 patients)

Figure 2.18.



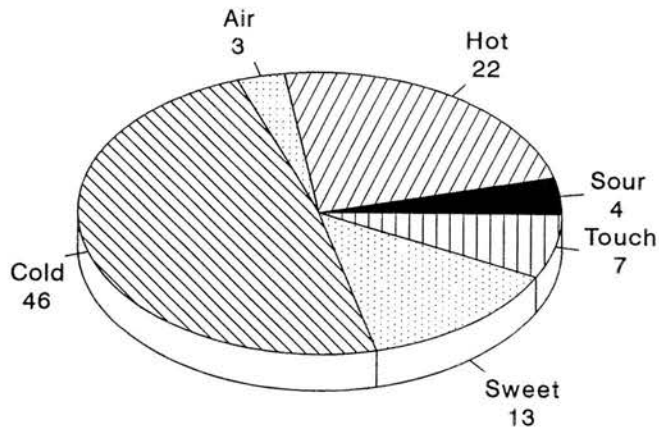
Proportion of test teeth responding to tactile stimulus (Yeaple probe)
Buccal surfaces (40 patients)

Figure 2.19.



Proportion of test teeth responding to thermal stimulus (cold air
blast) Buccal surfaces (40 patients)

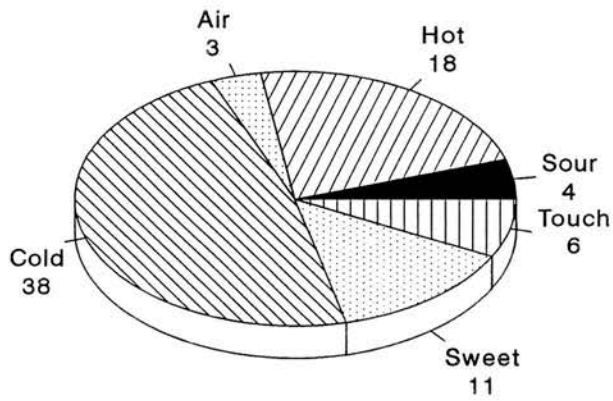
Figure 2.20.(a)



Screened Participants (n=49)

Reported frequency to various painful stimuli associated with cervical dentinal sensitivity (49 patients)

Figure 2.20.(b)



Trial Participants (n=40)

Reported frequency to various painful stimuli associated with cervical dentinal sensitivity (40 patients)

Yeaple probe calibration

Prior to each clinical session, the investigator calibrated the Yeaple probe using four mA readings (19, 45, 60, 72). The results are shown in **Table 2.4.a-d** and a calibration curve charted (**Fig. 2.21.**). There were no significant differences between pre-clinical and clinical calibration values.

Probe evaluation

Baseline scores for both tactile sensitivity to the Yeaple probe (**Table 2.5., Fig. 2.22.**) and Tactile VAS (**Table 2.6., Fig. 2.23**) were compared for the two groups and found to exhibit no significant differences. Mean probe scores for the silica-based group (test) (log-transformed) increased by 0.045 (3.9%), 0.12 (10.6%) and 0.26 (22.5%) in relation to the baseline scores over the 2-, 4-, and 8-week intervals respectively, indicating a decrease in sensitivity. The 2-week increase was not significant (95% C.I. for the ratio: 0.77 to 1.05), while those for the 4- and 8-week increases were significant (95% C.I. for the ratio: 0.60 to 0.95 and 0.42 to 0.74 respectively). For the diatomaceous earth group (control) the mean probe scores increased by 0.0084 (0.7%), 0.119 (10.5%), and 0.279 (24.6%) over the same time intervals. The 2-week increment again was not significant (95% C.I. for the ratio: 0.85 to 1.12), but the 4- and 8-week increments were significant (95% C.I. for the ratio: 0.60 to 0.96, and 0.41 to 0.67). Mean Tactile VAS scores (excluding outlier) for the silica-based group (test) decreased in relation to the baseline score by 0.90 (25.6%), 1.18 (33.6%), 1.62 (46.3%) over the 2-, 4-, and 8-week intervals respectively. The 2- and 8-week decreases were very highly significant (95% C.I. for the difference between the means: 0.55 to 1.53 and 0.83 to 2.42) while the 4-week decrease was highly significant (95% C.I.: 0.44 to 1.92). For the diatomaceous earth group (control) mean Tactile VAS scores decreased by 1.22 (35.1%), 1.47 (42.5%) and 1.68 (48.5%) respectively over the same time intervals. The 2- and 8-

week decreases were again very highly significant (95% C.I. for the difference between the means: 0.65 to 1.78, and 0.92 to 2.44) while the 4-week was highly significant (95% C.I.: 0.40 to 2.55). For both tactile sensitivity to probe and Tactile VAS scores, the results indicated a regular trend towards reduction in sensitivity with time, but without any apparent or detectable differences between the groups (Tables 2.5.-2.6., Figs. 2.22.-2.23.).

Cold Air Sensitivity

As with tactile sensitivity, air sensitivity values were indistinguishable between the groups at baseline (Table 2.7., Fig. 2.24.). Mean VAS scores for the test group decreased by 1.34 (25.4%), 1.68 (31.9%), and 2.55 (48.3%) over the three time intervals. All these decrements differed significantly from 0 (95% C.I.: 0.24 to 2.44, 0.59 to 2.78, and 1.41 to 3.70). For the control group the scores decreased by 1.04 (20.4%), 1.87 (36.6%) and 2.26 (44.3%) and again these values all differed significantly from baseline (95% C.I.: 0.45 to 1.63, 1.08 to 2.65 and 1.32 to 3.21). There were no inter-group significant differences at any time interval. These results again indicated a regular trend toward reduction in sensitivity to cold with time, but without any apparent or detectable differences between the groups (Table 2.7., Fig. 2.24.).

Subjective Evaluation - Overall Sensitivity VAS

Overall sensitivity VAS score values were indistinguishable between the groups at baseline (Table 2.8., Fig. 2.25.). Mean VAS scores for the test group decreased by 1.05 (25.2%), 1.69 (40.7%) and 2.21 (53.2%) over the three time intervals. All these decrements differed significantly from 0 (95% C.I.: 0.045 to 2.05, 0.64 to 2.74 and 1.10 to 3.32). For the control group the scores decreased by 1.14 (26.7%), 1.34 (30.2%), and 2.28 (51.4%) and again these values all differed significantly from 0 (95% C.I.: 0.34 to 1.94, 0.22 to 2.46 and 1.42 to 3.15). There were no intergroup significant differences at any time

interval. As with the other variables there was a regular trend towards reduction in sensitivity with time, but without any apparent difference between the groups (**Table 2.8.**, **Fig. 2.25.**).

Table 2.4.(a)

Calibration values (Yeaple probe gram weight) recorded prior to each clinical session during 8 week study (1989 - 1990)

mA Reading	19	45	60	72	19	45	60	72
gm*	10.0	30.0	50.0	70.0	10.0	30.0	50.0	70.0
	8.2	31.5	48.2	70.6	8.3	32.2	51.5	71.4
	8.9	31.6	50.7	70.7	9.1	32.5	51.6	71.5
	9.0	30.8	50.4	71.8	9.7	29.3	51.9	72.9
	10.3	30.1	49.2	70.3	9.9	29.0	49.1	68.3
	10.0	31.0	50.0	70.4	9.6	31.5	51.7	73.0
	7.8	30.2	50.6	70.6	8.4	28.0	49.2	69.5
	7.0	28.2	49.8	68.6	12.7	29.0	52.2	72.1
	9.8	29.6	49.4	69.0	9.8	31.9	50.4	70.4
	11.0	29.1	51.2	71.4	11.6	33.9	50.3	70.5
	10.3	30.3	51.7	71.1	10.6	30.6	52.6	72.5
	8.4	30.5	48.5	71.1	8.8	30.4	49.2	72.9
	10.0	30.8	52.6	71.6	11.0	30.0	49.0	71.2
	9.7	30.0	51.2	71.1	9.6	31.4	50.4	71.9
	10.0	28.9	51.5	69.9	8.6	30.3	51.8	70.5
	10.6	29.6	48.0	70.0	11.4	29.0	52.0	71.0
	9.8	30.2	52.7	70.6	10.4	30.0	49.3	72.0
	10.0	31.4	51.0	70.3	10.1	31.9	49.9	69.2
	9.8	29.2	51.7	71.7	9.4	31.6	51.1	70.8
	9.2	31.5	51.9	71.9	10.3	30.1	49.8	69.1
	9.4	31.4	50.1	71.3	9.0	30.6	50.4	70.6
	11.1	29.9	48.7	71.0	9.5	30.9	50.7	72.1
	10.6	28.8	52.2	70.2	10.2	31.6	52.0	68.8
	10.0	31.2	49.8	71.4	8.4	30.0	49.6	68.9
	11.4	30.4	49.6	70.4	8.2	30.7	49.1	71.6
	9.2	31.7	49.5	70.3	8.0	29.2	50.4	71.2
	9.6	28.6	51.1	69.8	10.1	32.5	51.5	70.0
	9.2	30.7	51.4	71.0	9.5	29.6	51.2	69.0
	8.1	30.2	50.5	71.3	7.8	29.0	50.9	69.2

Table 2.4.(a) cont.

mA Reading	19	45	60	72	19	45	60	72
gm*	10.0	30.0	50.0	70.0	10.0	30.0	50.0	70.0
	10.3	30.2	51.7	69.2	9.3	29.5	51.0	72.0
	11.4	28.9	50.2	71.4	9.7	29.7	50.6	70.9
	10.4	30.5	51.5	71.2	9.0	28.9	51.2	71.7
	9.1	29.9	51.5	69.3	10.6	30.4	50.6	70.1
	10.1	29.9	49.7	71.2	9.0	30.8	50.2	70.3
	11.2	30.7	51.0	70.1	8.6	31.2	50.4	70.8
	9.5	29.6	51.4	69.6	9.6	30.3	50.3	70.5
	11.0	29.2	50.6	71.2	10.0	30.8	49.0	69.0
	10.4	29.7	51.8	70.7	10.9	29.7	49.4	71.2
	9.8	29.4	50.8	69.0	9.0	29.2	51.6	71.0
	10.2	29.9	50.5	70.1	9.0	31.4	50.9	69.2
	9.9	29.0	48.9	69.1	11.6	30.6	49.2	71.1
	9.4	29.0	49.0	70.8	9.4	30.0	50.8	71.8
	11.0	31.0	51.6	71.2	9.0	29.3	49.7	70.5
	10.0	29.2	50.6	71.2	11.4	29.8	49.0	69.6
	9.2	31.2	48.8	70.2	9.0	30.3	50.9	70.1
	9.0	29.3	50.5	70.7	11.0	29.3	49.5	69.7
	9.0	30.2	49.5	67.4	9.7	30.4	48.6	70.0
	9.4	30.0	49.0	69.8	10.5	30.6	49.0	69.4
	9.8	30.6	49.2	69.5	11.0	30.9	50.0	69.0
	10.1	30.0	50.2	70.0	10.5	29.4	51.7	69.0
	9.1	31.0	50.5	69.7	9.4	29.3	51.8	71.8
	9.9	30.5	49.5	70.9	10.1	30.3	49.6	70.2
	9.2	29.2	50.5	69.5	9.1	30.9	50.7	69.5
	10.1	29.3	50.9	69.5	9.0	29.5	49.7	70.7
	9.3	29.3	49.6	71.0	10.8	31.8	51.2	70.6
	10.2	31.2	49.6	69.9	9.0	29.6	49.9	69.0
	9.9	29.7	50.5	69.1	10.5	30.8	50.3	69.3
	8.6	30.0	51.8	70.0	10.8	30.1	51.9	71.0

*gram weight

Table 2.4.(a) cont.

mA Reading	19	45	60	72	19	45	60	72
gm*	10.1	29.3	50.9	69.5	9.0	29.5	49.7	70.7
	9.3	29.3	49.6	71.0	10.8	31.8	51.2	70.6
	10.2	31.2	49.6	69.9	9.0	29.6	49.9	69.0
	10.0	30.2	51.8	71.2	9.3	31.4	49.6	70.6
	10.6	29.8	51.2	70.8	10.3	29.0	49.2	70.4
	10.0	30.4	50.6	70.4	9.3	29.4	50.4	71.0
	9.0	31.3	49.7	69.9	9.7	30.0	50.6	69.0
	10.9	29.6	49.4	69.2	10.8	30.6	50.2	69.5

*gram weight

Table 2.4.(b)

Calibration values (Yeaple probe gram weight) recorded prior to each clinical session at the 20 week time interval (1990)

mA Reading	19	45	60	72	19	45	60	72
gm*	10.0	30.0	50.0	70.0	10.0	30.0	50.0	70.0
	10.0	30.6	50.8	71.3	9.1	31.3	50.8	70.9
	10.4	28.8	49.9	71.8	10.3	29.1	50.8	69.0
	10.3	30.0	51.5	70.6	9.6	30.4	51.9	70.3
	9.4	31.5	51.2	71.0	9.9	30.5	50.5	70.6
	9.3	31.0	50.7	70.5	9.9	30.1	50.3	69.5
	9.2	29.7	49.6	70.7	10.4	31.0	49.4	71.6
	9.2	30.4	49.0	70.6	8.8	28.6	51.8	69.3
	9.2	31.4	50.4	69.8	9.6	29.6	49.7	69.2
	10.1	31.7	50.6	71.0	9.0	29.2	50.0	70.2
	9.5	30.0	51.6	70.2	10.2	29.2	49.1	69.8
	9.8	31.0	50.1	70.7	9.3	30.6	49.5	72.0
	8.8	31.7	50.8	70.0	9.0	30.0	50.6	69.4
	9.7	30.1	49.0	71.5	9.6	29.6	49.6	70.2
	10.0	31.0	49.9	68.3	9.7	31.2	50.2	70.3
	9.4	30.4	50.5	70.8	9.4	31.0	50.3	70.0
	10.0	30.6	50.1	71.0	9.1	30.4	50.0	69.8
	9.2	30.2	50.9	70.2				

Table 2.4.(c)

Summary: Calibration values (Yeaple probe gram weight) recorded prior to each clinical session during the 8-week clinical study

mA	19	45	60	72
gm*	9.8	30.3	50.4	70.4
SD	0.933	1.274	1.053	1.040
SEM	0.838	0.114	0.095	0.093

*gram weight

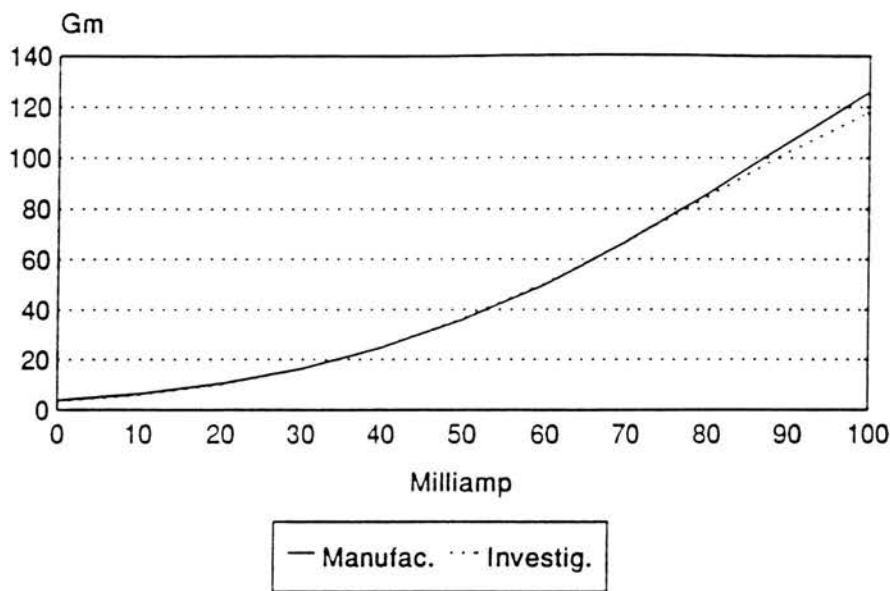
Table 2.4.(d)

Summary: Calibration values (Yeaple probe gram weight) recorded prior to each clinical session at the 20 week time interval

mA	19	45	60	72
gm*	9.6	30.3	50.3	70.4
SD	0.465	0.805	0.746	0.812
SEM	0.079	0.136	0.126	0.137

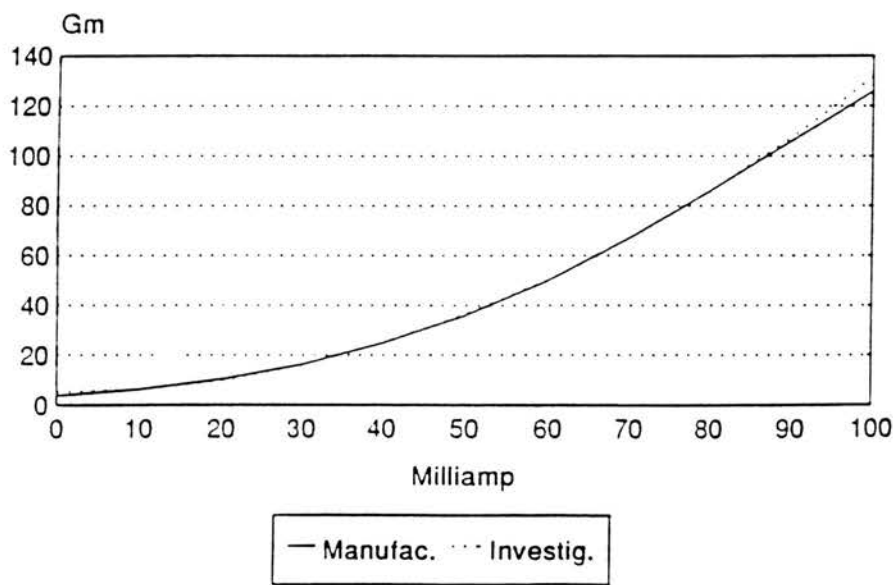
*gram weight

Figure 2.21.(a)



Yeaple probe calibration mean values (gram weight) recorded prior to each clinical session during the 8 week study

Figure 2.21.(b)



Yeaple probe calibration mean values (gram weight) recorded prior to each clinical session at the 20 week time interval

Table 2.5.(a)

Comparison of Yeaple probe (Log_{10} transformed) mean scores (gram weight) for test and control groups during the 8 and 20 week study

Time	Gp	N	Mean	SD	SEM
Baseline	Test	20	1.14	0.133	0.030
	Control	20	1.13	0.157	0.035
2-week	Test	20	1.19	0.153	0.034
	Control	20	1.14	0.169	0.038
4-week	Test	20	1.26	0.200	0.045
	Control	20	1.25	0.285	0.064
8-week	Test	20	1.40	0.257	0.057
	Control	20	1.41	0.275	0.062
20-week	Test	20	1.32	0.239	0.053
	Control	20	1.34	0.254	0.057
B-2 wk	Test	20	-0.045	0.149	0.033
	Control	20	-0.008	0.127	0.028
B-4 wk	Test	20	-0.121	0.214	0.048
	Control	20	-0.119	0.219	0.049
B-8 wk	Test	20	-0.256	0.263	0.059
	Control	20	-0.279	0.229	0.051
B-20 wk	Test	20	-0.176	0.228	0.051
	Control	20	-0.207	0.267	0.060

Test = Silica-based group

Control = Diatomaceous earth group

Table 2.5.(b)

Comparison of Yeaple probe (Untransformed) mean scores (gram weight) between test and control groups during the 8 and 20 week study

Time	Gp	N	Mean	SD	SEM
Baseline	Test	20	14.56	5.070	1.134
	Control	20	14.71	7.423	1.660
2-week	Test	20	16.36	6.255	1.399
	Control	20	15.00	6.438	1.440
4-week	Test	20	20.38	9.910	2.216
	Control	20	22.88	19.572	4.377
8-week	Test	20	29.38	16.797	3.756
	Control	20	31.13	19.065	4.263
20-week	Test	20	24.0	13.064	2.921
	Control	20	25.88	15.734	3.518
B-2 wk	Test	20	-1.81	5.418	1.212
	Control	20	-0.29	4.984	1.115
B-4 wk	Test	20	-5.81	10.010	2.238
	Control	20	-8.17	16.120	3.605
B-8 wk	Test	20	-14.81	17.088	3.821
	Control	20	-16.42	15.936	3.563
B-20 wk	Test	20	-9.44	11.862	2.653
	Control	20	-11.17	16.611	3.714

Test = Silica-based group

Control = Diatomaceous earth group

Table 2.5.(c)

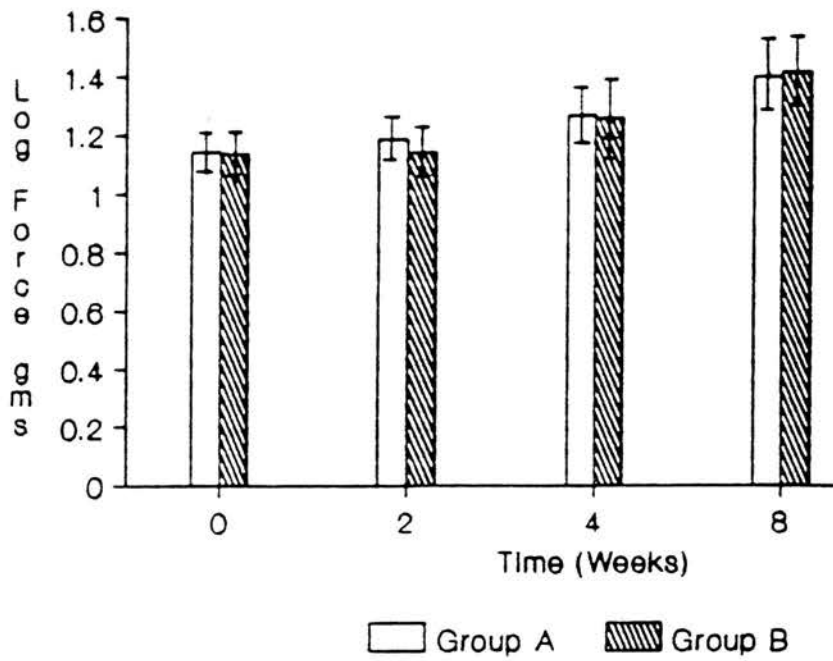
Summary: Comparison of response to Tactile stimulus (Yeaple probe - gram weight) between test and control groups during the 8 week study (Log₁₀ transformed data).

Time	Test Gp (n = 20) Mean (SD)	% Change	Control Gp (n = 20) Mean (SD)	% Change
Baseline	1.14 (0.133)	0	1.13 (0.157)	0
2-Weeks	1.19 (0.153)	-3.9%	1.14 (0.169)	-0.7%
4-Weeks	1.26 (0.200)	-10.6%	1.25 (0.285)	-10.5%
8-Weeks	1.40 (0.257)	-22.5%	1.41 (0.275)	-24.6%

Test = Silica-based group

Control = Diatomaceous earth group

Figure 2.22.



Group A (Test) = Silica-based group
Group B (control) = Diatomaceous earth group

Comparison of Yeaple probe (Log_{10} transformed) mean scores (gram weight) for test and control groups during the 8 week study (Including 95% Confidence Intervals)

Table 2.6.(a)

Comparison of mean Yeaple probe (VAS) scores (cm) between test and control groups during the 8 and 20 week study

Time	Gp	N	Mean	SD	SEM
Baseline	Test	20	3.51	1.593	0.356
	Control	20	3.46	1.793	0.401
2-week	Test	20	2.61	1.361	0.312*
	Control	20	2.25	1.381	0.309
4-week	Test	20	2.33	1.732	0.387
	Control	20	1.99	1.746	0.390
8-week	Test	20	1.88	1.569	0.3518
	Control	20	1.78	1.273	0.285
20-week	Test	20	2.35	1.633	0.365
	Control	20	1.75	1.533	0.343
B-2 wk	Test	20	1.04	1.018	0.233*
	Control	20	1.22	1.208	0.270
B-4 wk	Test	20	1.17	1.584	0.354
	Control	20	1.47	2.296	0.513
B-8 wk	Test	20	1.62	1.696	0.379
	Control	20	1.68	1.623	0.363
B-20 wk	Test	20	1.15	1.341	0.300
	Control	20	1.71	2.040	0.456

* One outlier eliminated from 2-week data for test group

Test = Silica-based group

Control = Diatomaceous earth group

Table 2.6.(b)

Summary: Comparison of mean Yeaple probe (VAS) scores (cm) between test and control groups during the 8 week study

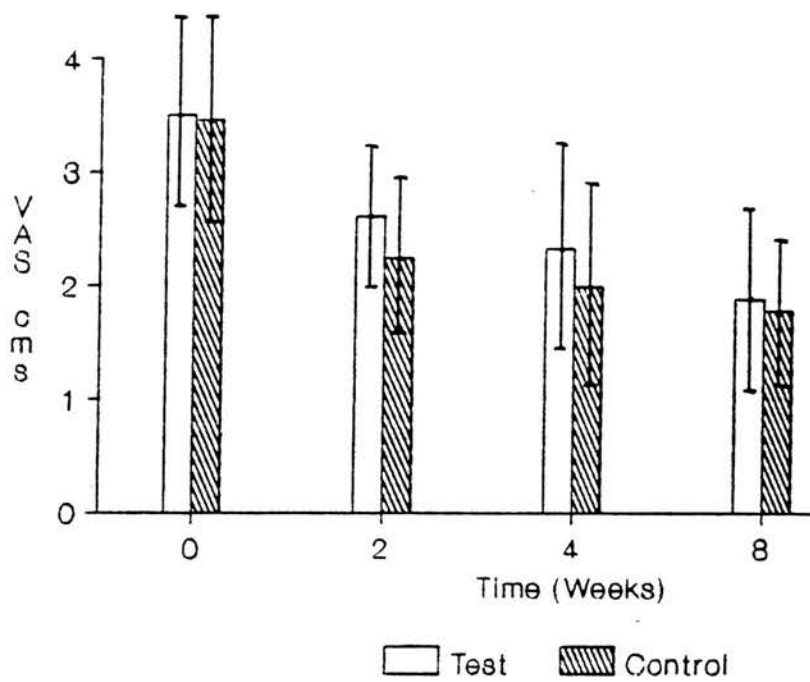
Time	Test Gp (n = 20) Mean (SD)	% Change	Control Gp (n = 20) Mean (SD)	% Change
Baseline	3.5 (1.59)	0	3.5 (1.79)	0
2-Weeks*	2.6 (1.36)	-25.6%	2.3 (1.38)	-35.1%
4-Weeks	2.3 (1.73)	-33.6%	2.0 (1.75)	-42.5%
8-Weeks	1.9 (1.57)	-46.3%	1.8 (1.27)	-48.5%

* one outlier eliminated from 2-week data for Test group

Test = Silica-based group

Control = Diatomaceous earth group

Figure 2.23.



Group A (Test) = Silica-based group
Group B (control) = Diatomaceous earth group

Comparison of mean Yeaple probe (VAS) scores (cm) between test and control groups during the 8 week study (Including 95% Confidence Intervals)

Table 2.7.(a)

Comparison of mean cold air blast (VAS) scores (cm) between test and control groups during the 8 and 20 week study

Time	Gp	N	Mean	SD	SEM
Baseline	Test	20	5.29	1.271	0.284
	Control	20	5.12	1.177	0.263
2-week	Test	20	3.95	1.965	0.440
	Control	20	4.07	1.910	0.427
4-week	Test	20	3.60	2.255	0.504
	Control	20	3.25	1.788	0.400
8-week	Test	20	2.73	2.265	0.507
	Control	20	2.85	2.564	0.573
20-week	Test	20	3.29	2.022	0.452
	Control	20	3.22	2.270	0.508
B-2 wk	Test	20	1.34	2.342	0.524
	Control	20	1.04	1.260	0.282
B-4 wk	Test	20	1.69	2.342	0.524
	Control	20	1.87	1.677	0.375
B-8 wk	Test	20	2.56	2.438	0.545
	Control	20	2.27	2.011	0.450
B-20 wk	Test	20	1.99	2.176	0.486
	Control	20	1.90	1.779	0.398

Test = Silica-based group

Control = Diatomaceous earth group

Table 2.7.(b)

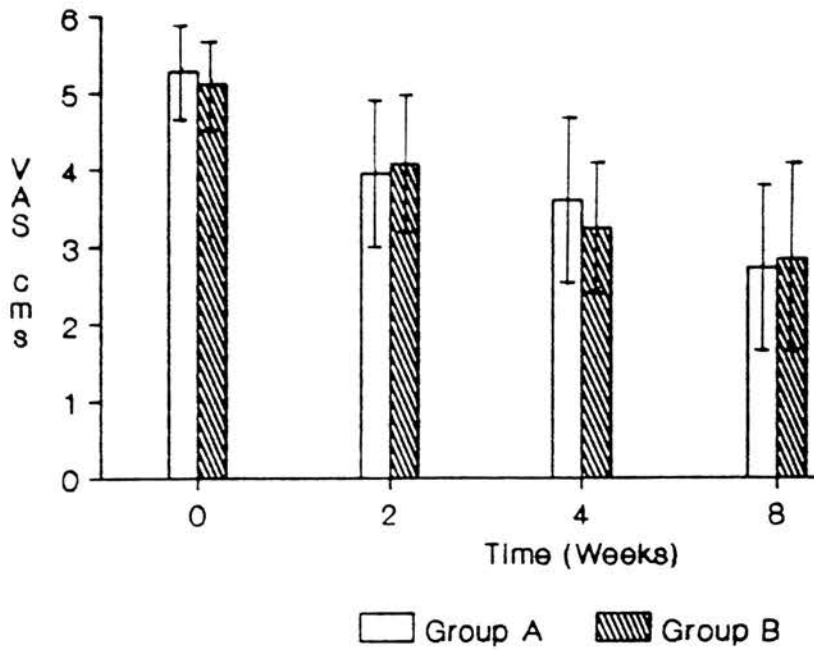
Summary: Comparison of mean VAS scores (cold air Blast - cm)
between test and control groups during the 8 week study

Time	Test Gp (n = 20) Mean (SD)	% Change	Control Gp (n = 20) Mean (SD)	% Change
Baseline	5.3 (1.27)	0	5.1 (1.18)	0
2-Weeks	3.9 (1.97)	-25.4%	4.1 (1.91)	-20.4%
4-Weeks	3.6 (2.26)	-31.9%	3.2 (1.79)	-36.6%
8-Weeks	2.7 (2.27)	-48.3%	2.9 (2.56)	-44.3%

Test = Silica-based group

Control = Diatomaceous earth group

Figure 2.24.



Group A (Test) = Silica-based group
Group B (Control) = Diatomaceous earth group

Comparison of mean VAS scores (cold air blast - cm) between test and control groups during the 8 week study (Including 95% Confidence Intervals)

Table 2.8.(a)

Comparison of mean Overall Sensitivity VAS scores (cm) between test and control groups during the 8 and 20 week study

Time	Gp	N	Mean	SD	SEM
Baseline	Test	20	4.15	1.921	0.430
	Control	20	4.44	2.027	0.453
2-wk	Test	20	3.11	2.304	0.515
	Control	20	3.31	2.087	0.467
4-wk	Test	20	2.46	1.943	0.434
	Control	20	3.11	2.140	0.478
8-wk	Test	20	1.94	1.935	0.433
	Control	20	2.16	1.963	0.439
20-wk	Test	20	2.86	2.859	0.639
	Control	20	2.43	2.317	0.518
B-2 wk	Test	20	1.05	2.141	0.479
	Control	20	1.14	1.700	0.380
B-4 wk	Test	20	1.69	2.240	0.501
	Control	20	1.34	2.392	0.535
B-8 wk	Test	20	2.21	2.370	0.530
	Control	20	2.29	1.840	0.411
B-20 wk	Test	20	1.29	2.490	0.557
	Control	20	2.02	2.380	0.532

Test = Silica-based group
Control = Diatomaceous earth group

Table 2.8.(b)

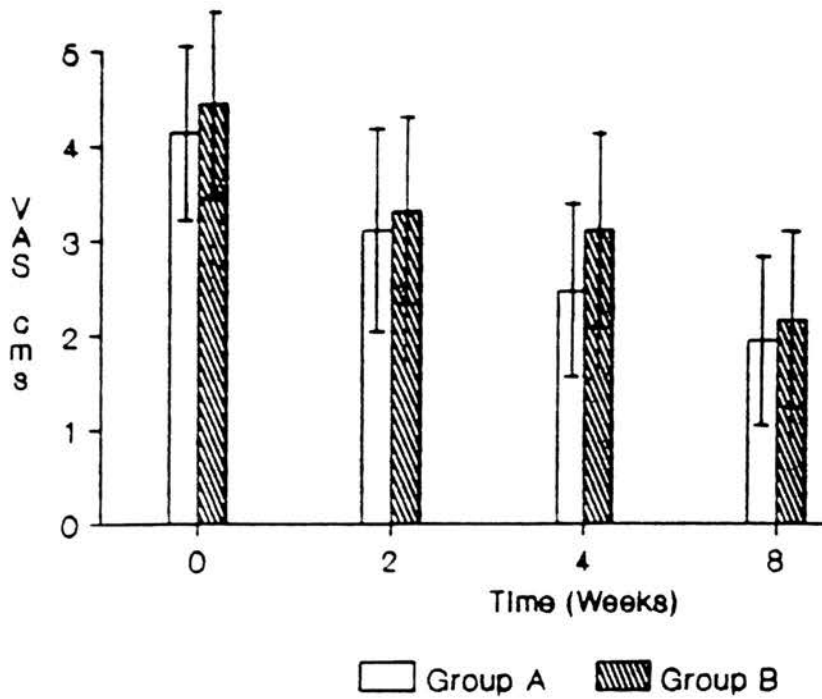
Summary: Comparison of mean VAS scores (cm) for Overall Sensitivity between test and control groups during the 8 week study

Time	Test Gp (n = 20) Mean (SD)	% Change	Control Gp (n = 20) Mean (SD)	% Change
Baseline	4.2 (1.92)	0	4.4 (2.03)	0
2-Weeks	3.1 (2.30)	-25.2%	3.3 (2.09)	-26.7%
4-Weeks	2.5 (1.94)	-40.7%	3.1 (2.14)	-30.2%
8-Weeks	1.9 (1.93)	-53.2%	2.2 (1.96)	-51.4%

Test = Silica-based group

Control = Diatomaceous earth group

Figure 2.25.



Group A (Test) = Silica-based group
Group B (Control) = Diatomaceous earth group

Comparison of mean VAS scores (cm) for Overall Sensitivity between test and control groups during the 8 week study (Including 95% Confidence Intervals)

Discussion

The two SCH dentifrices used in this study were similar except for their respective abrasive systems (silica-based and diatomaceous earth). If the reduction in sensitivity levels was attributed to the silica-based component as suggested by Addy *et al.* (1987b), then one would have expected the group using the precipitated silica to have demonstrated a significant difference in reduction of sensitivity. The results of this study, however, demonstrate that the two SCH dentifrices were equally effective (**Tables 2.5.-2.8., Figs.2.22.-2.25.**) and would appear to confirm McCall & Hamrick's conclusions in respect to the role of the abrasive component. It is recognised, however, that the precipitated silicas used in the various studies may not be the same and this, together with the difference in formulation may have resulted in the reported observations by the investigators. These results also highlight the discrepancy which may be observed between laboratory and clinical findings, which may not necessarily favour the former.

One of the problems associated with studies of this type is the influence of the placebo effect where participants using control pastes with no active ingredients may experience significant reductions in their sensitivity levels (this effect would also be present in the test group). The placebo and Hawthorne effect together with the recognised non-placebo effects, e.g., an improvement in the participants oral hygiene (Peden 1977) and possible natural desensitization in time (Karlson & Penney 1975) may also contribute to a reduction in sensitivity levels.

While a placebo effect was possible in this study, the study was randomised and double-blind and the patients were in no way informed in a manner which would have implied efficacy for either dentifrice. The absence of a placebo group in this study may, however, be criticised, although it should be acknowledged that this study was primarily concerned with the problem of abrasivity (i.e., the abrasive component) and as such the diatomaceous-earth SCH dentifrice was used

as a positive control. Further, SCH dentifrices have been shown to produce significantly greater reduction in sensitivity levels than a placebo using a similar range of assessment methods (Minkoff & Axelrod 1987). On a more practical note the addition of a placebo group would have increased patient recruitment to 60 patients. In any event, it may be considered doubtful practice to refuse treatment for patients suffering pain, any more for the absence of a fluoride dentifrice in a caries study evaluating a new dentifrice. This problem may highlight the difficulties in testing a new desensitizing agent in that at present there appears to be no ideal positive or negative controls despite claims to the contrary.

Although criticism of the absence of any form of stratification in the study design may be valid, in that this could lead to an imbalance in the groups, it can be pointed out that for all variables (age, sex, sensitivity levels etc), both groups appeared to be balanced at baseline (**Tables 2.1., 2.5.-2.8.**).

The results of this randomised double-blind parallel study of 40 patients with CDS over 8 weeks of product use demonstrated that when assessed with tactile and cold air stimuli, together with patient subjective response, the SCH dentifrices were equally effective and seemed to act to the same degree in relation to time. The response to both dentifrices was evident within 4 weeks of use and the degree of improvement increased during the duration of the 8-week study. In conclusion, the results of this study suggest that changing the abrasive component of SCH dentifrices did not significantly increase or decrease the desensitizing activity of the original product.

CHAPTER 3

Results 12 weeks following cessation of 8 weeks supervised dentifrice use

Introduction

SCH has been widely used in a dentifrice form for the treatment of CDS (**section 1.4.3.4.**). Concern, however, has been expressed with regard to the lack of information about quantification of the test stimuli under suitably controlled conditions, as well as to the absence of an objective method for evaluating dentifrice effect in reducing CDS (Council on Dental Therapeutics 1985). These deficiencies were addressed in a 12 week, double-blind, parallel comparative (placebo) study (Minkoff & Axelrod 1987), in which levels of sensitivity in affected teeth were assessed by 3 methods, thermally controlled cold air stimulus, tactile stimulus with an electronic pressure-sensitive probe (Yeaple probe), and subjective response. The authors concluded that the results from all 3 methods of assessment indicated that SCH was significantly more effective than a placebo in reducing CDS.

The effectiveness of the active ingredient (SCH) has previously been questioned, with several investigators attributing any observed reduction in CDS to the abrasive component of the dentifrice (**section 1.4.4.22.**). Several studies (Manochehr-Pour et al. 1984, Silverman 1985, McFall & Hamrick 1987, Addy et al. 1987b, Salvato et al. 1989, Jackson et al. 1989, 1990) have utilised a low abrasive component in desensitizing dentifrices with varying results. Some of these investigators (Addy et al. 1987b, Jackson et al. 1989, 1990) reported that a silica-based product containing SrAC₂F was more effective than SCH with the abrasive diatomaceous earth.

Few clinical studies based on the Council on Dental Therapeutics (1985) recommendations for objective as well as subjective methods for evaluating dentifrice effects have reported any follow-up data following cessation of dentifrice use.

The purpose of this study was to provide such data, based on 3

accepted methods of assessment, following cessation of controlled SCH dentifrice use.

3.1. Materials and Methods

During the original 8-week clinical 2-way comparative parallel study of 40 patients, a non-commercially available SCH dentifrice with a silica-based abrasive was compared with a commercially available SCH dentifrice containing the abrasive diatomaceous earth (Sensodyne). Both dentifrices were closely matched with respect to taste, colour, consistency, and appearance. All 40 patients returned to be reexamined at the 20-week point (**Table 2.1.**). During the follow-up examination, the assessment procedures were as in the main 8-week clinical study (**section 2.2.4.**).

3.1.1. Data Analysis

All data were tested for normality by plotting in ascending order of magnitude against the corresponding normal scores.

All proved to be normally distributed with the exception of Tactile Force, which was then normalized by means of logarithmic transformation. Data analysis was complicated by the fact that, since the readings were time-dependent, it was not possible to undertake a straightforward multiple regression or analysis of variance. To avoid this, the 4 main sources of data, Tactile Force, Tactile VAS, Cold Air Sensitivity, and Overall Sensitivity VAS, were analysed independently using the following procedures:

1. Inter-group and within-group comparisons of change in response from baseline to 20 weeks. If this within-group test proved significant then,
2. Inter-group comparison of the rate of change within the study time period.
3. Inter-group comparison of the overall level of response within

the time period.

Of these, 1) was achieved by comparing the differences between group mean scores at baseline and at 20 weeks using firstly a paired t-test (19 degrees of freedom) to see if there was a significant difference within each group, and secondly an unpaired t-test (38 degrees of freedom) to measure any relative differences between groups.

Analysis of 2) was accomplished by calculating a regression coefficient for each patient in both groups for the total time period (readings at 0, 2, 4, 8, and 20 weeks). The mean regression coefficients for each group were then compared using an unpaired t-test (38 degrees of freedom).

Finally, the mean scores of the 5 timed readings at each time point for each patient were computed and the 2 group means compared, again using unpaired t-tests (38 degrees of freedom) to ensure no bias existed between groups in terms of the proportion of high or low responses within each group.

3.2. Results

No changes were observed in the oral tissues of any patient in either group following cessation of the clinical study. Twenty four patients received no dental treatment during the 12-week post-completion period. Of the remaining 16, 9 received scaling and polishing, 4 had teeth restored, 2 had an examination only, and 1 received penicillin for a periodontal abscess. Treatment did not involve any of the study teeth.

As patients had not been advised there would be a recall visit at the time of the original study, no reliable information was available concerning subsequent dentifrice use.

The results for the original 8-week clinical study are summarized in **Tables 2.5.-2.8..** The results for all variables indicated a remarkably regular trend towards reduction with time but without any apparent differences between the silica-based (low abrasive) and diatomaceous earth groups. The results for the 20-week study are shown in **Tables 3.1.-3.4., Figs. 3.1.-3.4..**

Table 3.1.

Summary: Comparison of Yeaple probe (Log_{10} transformed data) mean scores (gram weight) for test and control groups during the 20 week study

Time	Test Gp (n = 20) Mean (SD)	% Change	Control Gp (n = 20) Mean (SD)	% Change
Baseline	1.14 (0.133)	0	1.13 (0.157)	0
8-Weeks	1.40 (0.257)	-22.5%	1.41 (0.275)	-24.6%
20-Weeks	1.32 (0.239)	-15.4%	1.34 (0.254)	-18.2%

Test = Silica-based group

Control = Diatomaceous earth group

Figure 3.1. (a)

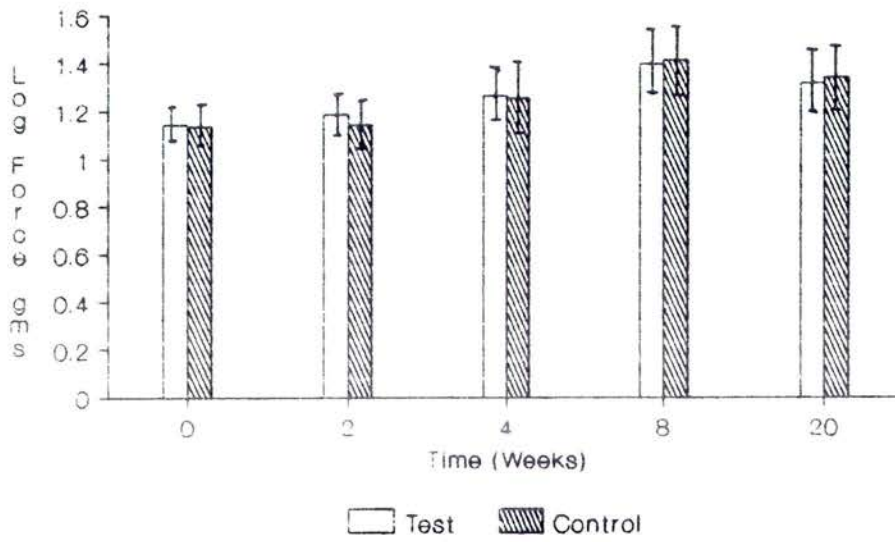
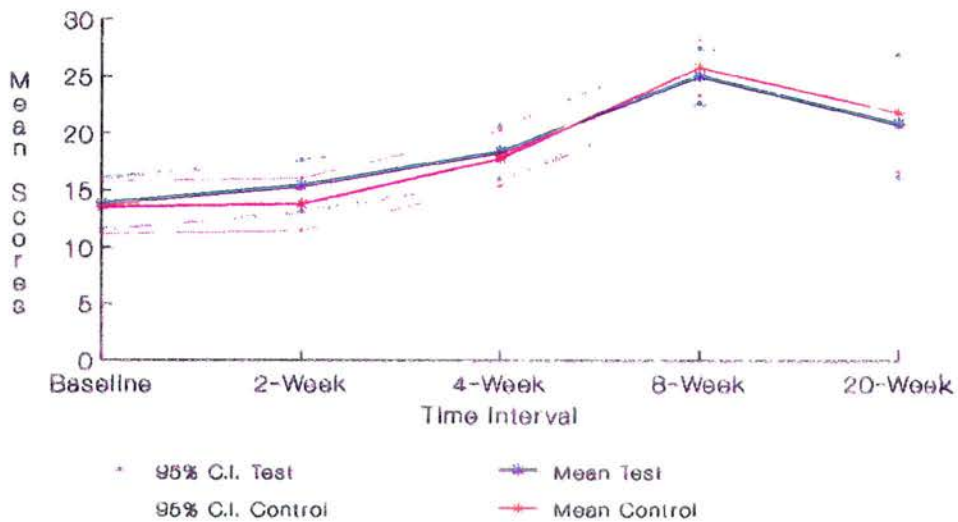


Figure 3.1. (b)



Test = Silica-based group
Control = Diatomaceous earth group

Comparison of Yeaple probe (Log₁₀ transformed) mean scores (gram weight) for test and control groups during the 20 week study (Including 95% Confidence Intervals)

Table 3.2.

Summary: Comparison of mean Yeaple probe (VAS) scores (cm) between test and control groups during the 20 week study

Time	Test Gp (n = 20) Mean (SD)	% Change	Control Gp (n = 20) Mean (SD)	% Change
Baseline	3.5 (1.59)	0	3.5 (1.79)	0
8-Weeks	1.9 (1.57)	-46.3%	1.8 (1.27)	-48.5%
20-Weeks	2.4 (1.63)	-32.8%	1.8 (1.53)	-49.3%

Test = Silica-based group

Control = Diatomaceous earth group

Figure 3.2.(a)

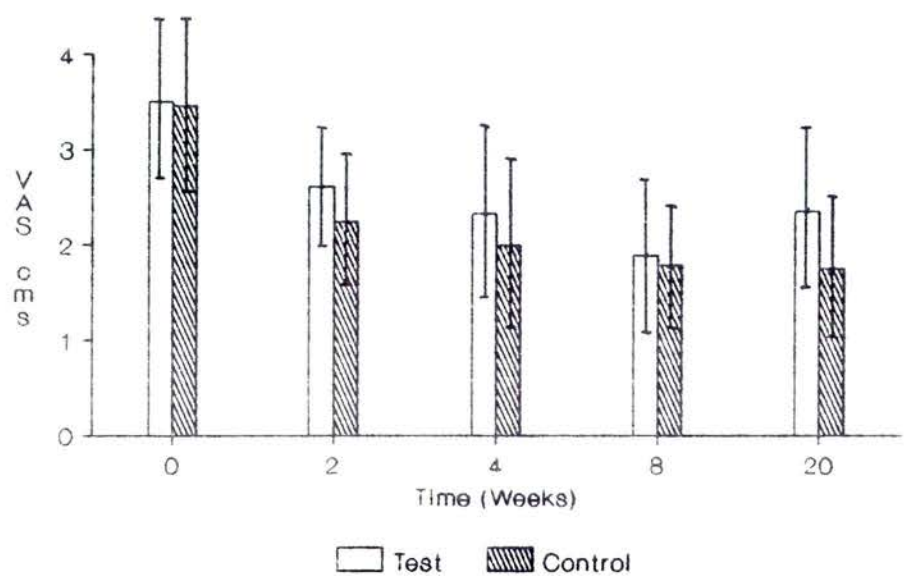
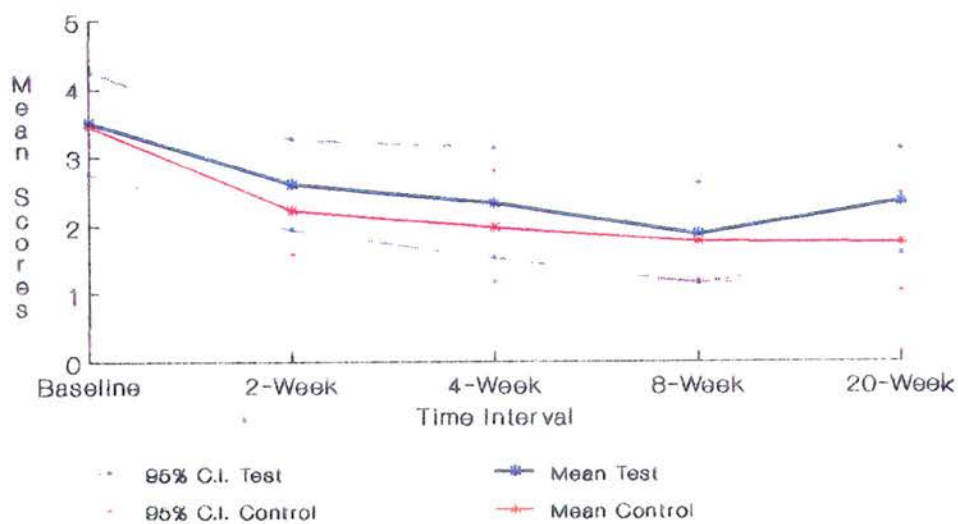


Figure 3.2.(b)



Test = Silica-based group
Control = Diatomaceous earth group

Comparison of mean Yeaple probe (VAS) scores (cm) between test and control groups during the 20 week study (Including 95% Confidence Intervals)

Table 3.3.

Summary: Comparison of mean cold air blast (VAS) scores (cm) between test and control groups during the 20 week study

Time	Test Gp (n = 20) Mean (SD)	% Change	Control GP (n = 20) Mean (SD)	% Change
Baseline	5.3 (1.27)	0	5.1 (1.18)	0
8-Weeks	2.7 (2.27)	-48.3%	2.9 (2.56)	-44.3%
20-Weeks	3.3 (2.02)	-37.7%	3.2 (2.27)	-37.0%

Test = Silica-based group

Control = Diatomaceous earth group

Figure 3.3.(a)

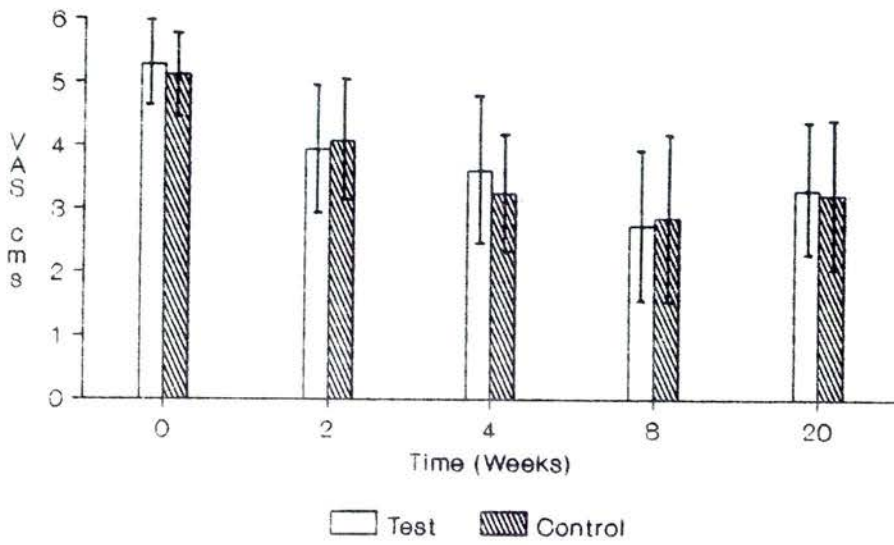
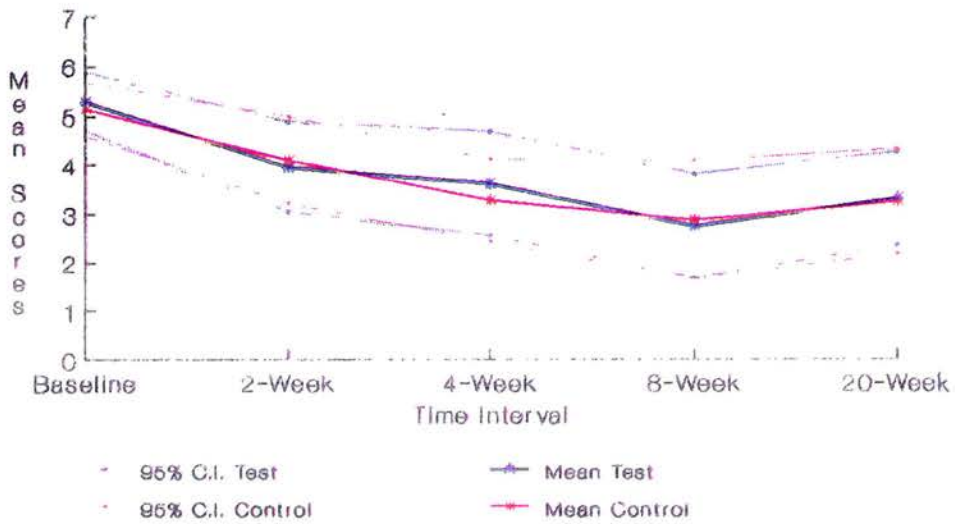


Figure 3.3.(b)



Test = Silica-based group
 Control = Diatomaceous earth group

Comparison of mean VAS scores (cold air blast - cm) between test and control groups during the 20 week study (Including 95% Confidence Intervals)

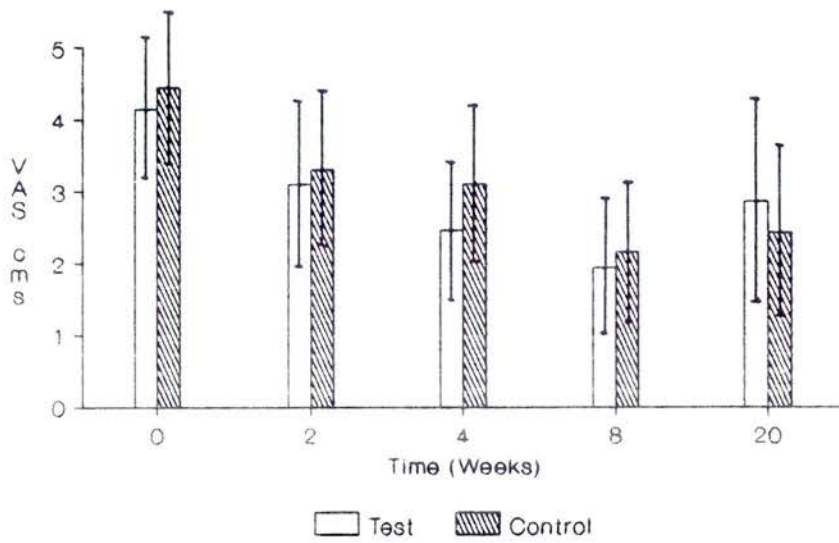
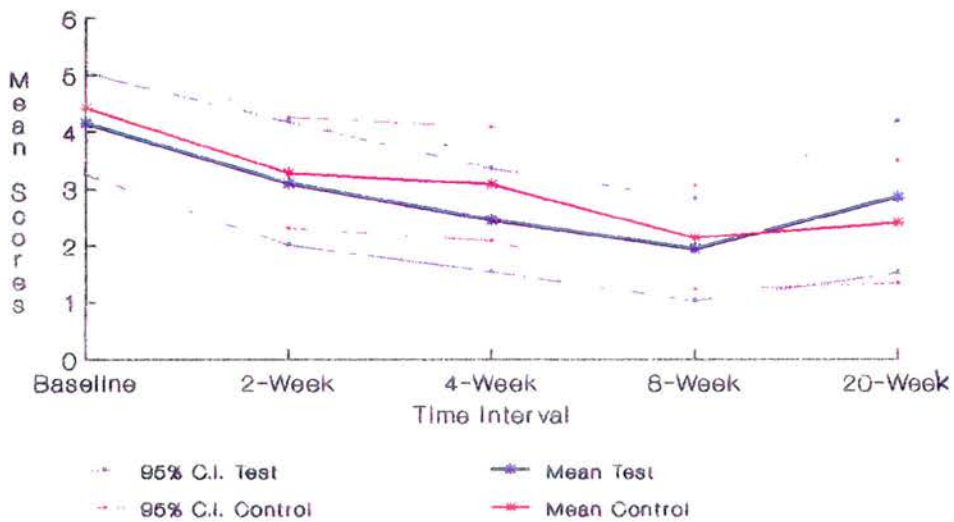
Table 3.4.

Summary: Comparison of mean Overall Sensitivity VAS scores (cm) between test and control groups during the 20 week study

Time	Test Gp (n = 20) Mean (SD)	% Change	Control Gp (n = 20) Mean (SD)	% Change
Baseline	4.2 (1.92)	0	4.4 (2.03)	0
8-Weeks	1.9 (1.93)	-53.2%	2.2 (1.96)	-51.4%
20-Weeks	2.9 (2.86)	-31.1%	2.4 (2.32)	-45.3%

Test = Silica-based group

Control = Diatomaceous earth group

Figure 3.4.(a)**Figure 3.4.(b)**

Test = Silica-based group
 Control = Diatomaceous earth group

Comparison of mean VAS scores (cm) for Overall Sensitivity between test and control groups during the 20 week study (Including 95% Confidence Intervals)

Discussion

Few studies have published data following cessation of dentifrice use. Addy *et al.* (1987b) reported a reversal in sensitivity levels towards baseline following cessation. In the 8-week study, the results for all variables showed a remarkably regular trend towards reduction with time (**Tables 3.1.-3.4., Figs. 3.1.-3.4.**). Following cessation of product use, it was found that for all variables there was a very slight and not statistically significant change in mean sensitivity levels in both dentifrice groups over the 12-week post-treatment period, with neither group showing a significantly higher or lower change compared with the other. The 95% Confidence Intervals (**Tables 3.1.-3.4.**) give minimum and maximum ranges for the mean expected differences likely to be seen in the population from which the samples are drawn. There is less than a 5% chance that the true population mean will lie outside this range. There were no significant differences in the rate of change of response between groups over the 12 week post-treatment period, and no significant differences in the overall level of response.

Although there was a very slight reversal of the trend demonstrated during the 8-week study, no apparent differences were detected between the two groups at the 20-week time interval and overall, sensitivity levels remained lower than at baseline.

Reductions in sensitivity levels achieved by both dentifrices were still evident three months after cessation of regular controlled use, changing the abrasive system did not appear to affect the desensitizing activity of the dentifrices.

The observation that there appeared to be a sustained reduction in sensitivity levels in both groups following cessation of controlled use may be of great importance particularly when recruiting patients for a study designed to test the efficacy of a desensitizing agent. Patients who are using a desensitizing dentifrice should be asked to refrain from using this paste and a substitute fluoride dentifrice should be provided for a specific period prior to the commencement of the study.

If this is not done, there is a real possibility that a carry over effect of the patient's existing dentifrice may effect the results of the intended study. In the original 8-week study, patients who were using a desensitizing dentifrice at the time of screening were placed on a substitute fluoride dentifrice for at least two months prior to inclusion into the study. The results of the 20-week study would appear to justify this action, although it should be stated that if any patient complained of increasing sensitivity following usage of the fluoride dentifrice, prospective involvement in the study would be terminated and advice would be given to revert back to using the original desensitizing dentifrice. No problems of this nature were reported to the investigator prior to the original 8-week study.

There may, however, be ethical and practical reasons against instituting a 2/3 month delay prior to the commencement of a study particularly in patients who are suffering discomfort from CDS.

It was concluded from this study that reductions in sensitivity levels achieved by the use of both dentifrices were still evident 3 months after the cessation of their regular controlled use, and that the abrasivity of the dentifrice did not affect its desensitizing activity.

CHAPTER 4THE EFFECT OF SCH DENTIFRICES ON PLAQUE
AND GINGIVAL CONDITIONIntroduction

Several investigators have suggested that plaque may play a role in the aetiology of CDS. Other work, however, indicates that plaque is not a significant aetiological factor in CDS, although several investigators stress the importance of good oral hygiene in its management (See pages xxi-xxii).

Recently Wallace & Bissada (1990) have suggested that plaque accumulation is associated with CDS, although in reviewing the available literature it would appear that patients who complain of CDS generally have a very high standard of oral hygiene, particularly at those sites where CDS is diagnosed as being present (Addy et al. 1987c, Addy 1992).

According to Addy et al. 1990b there is, however, a lack of information with regard to the effect(s) of desensitizing dentifrices on oral hygiene. Furthermore, according to these investigators, most of the earlier studies appeared to have based their findings (e.g., beneficial effects of the dentifrice) on the purely subjective evaluation of improved oral hygiene without actually recording the differences in plaque scores between the dentifrices.

Although SCH dentifrices have been widely used for the treatment of CDS (**section 1.4.3.4.**), there have been only a few studies of their effect on plaque. Several investigators have claimed that silica-based products containing SrAc₂F were more effective in reducing plaque than a SCH dentifrice containing the abrasive diatomaceous earth (Jackson et al. 1989, Addy et al. 1990b). The purpose of this study, therefore, was to evaluate whether levels of plaque and gingival inflammation were affected by 2 desensitivity dentifrices differing only in their abrasivity.

4.1. Materials and Methods

4.1.1. Reproducibility Study

Prior to the main 8-week clinical study, the investigator assessed both plaque and gingival condition in 2 patients (324 sites) for the purposes of reproducibility.

Plaque accumulation

The investigator assessed the amount of plaque present on all teeth using the Silness and Løe Index (1964). Assessment was from six sites; mesio-buccal, mid-buccal, disto-buccal, disto-lingual, mid-lingual, and mesio-lingual on teeth 1-7 in each quadrant. The initial plaque scores were recorded by an assistant on the appropriate clinical form (**Fig. 4.1.**).

Gingival condition

The investigator also assessed the gingival condition using the Løe and Silness Gingival Index (1963) at the same six sites on teeth 1-7 in each quadrant. The initial scores were recorded by an assistant on the appropriate clinical form (**Fig. 4.2.**). A second measurement of both plaque and gingival condition was recorded approximately 30 minutes after the initial examination.

4.1.2. Clinical study

Forty patients, 15 male and 25 female, mean age 42.8 (SD 8.2) years participated in the study (**Table 2.1.**), the details of which have been previously described (**Chapter 2**).

Plaque was assessed using the Silness and Løe Index (1964) and gingival condition by the Løe and Silness Gingival Index (1963). Both indices were determined at six sites; mesio-buccal, mid-buccal, disto-buccal, disto-lingual, mid-lingual and mesio-lingual on teeth 1-7 in each quadrant at 1 week pre-baseline, baseline, and at 2, 4 and 8 weeks thereafter.

Figure 4.1.

UPPER TEETH	7	6	5	RIGHT			4	3	2	1	D M	M D	1	2	3	4	LEFT			5	6	7
Buccal																						
Palatal																						
\bar{x}																						
Average																						

LOWER TEETH	7	6	5	RIGHT			4	3	2	1	D M	M D	1	2	3	4	LEFT			5	6	7
Lingual																						
Buccal																						
\bar{x}																						
Average																						

Plaque Index form

Figure 4.2.

UPPER TEETH	7	6	5	RIGHT			4	3	2	1	D M	M D	1	2	3	4	LEFT			5	6	7
Buccal																						
Palatal																						
\bar{x}																						
Average																						

LOWER TEETH	7	6	5	RIGHT			4	3	2	1	D M	M D	1	2	3	4	LEFT			5	6	7
Lingual																						
Buccal																						
\bar{x}																						
Average																						

Gingival Index form

4.1.3. Data Analysis

Reproducibility Study

Cohen's Kappa (1960) and percentage reproducibility tests were used to assess scores for both plaque and gingival condition. The Kappa statistic relates the actual measurement of agreement obtained with the degree of agreement which would have been obtained had the diagnoses been made at random or, in other words, the extent to which the actual degree of agreement recorded improves upon chance. This is probably the most reliable way of assessing overall examiner agreement between the two visits. Both unweighted and weighted kappa were used since unweighted kappa only considers area of total agreement, it takes no account of 'near misses'. Applying a weighted system to such scores ensures that they can make an appropriate and realistic contribution to the kappa statistic.

Dice's Coincidence Index was also utilised, providing a measure of either the probability that a tooth (or surface) diagnosed as normal by the examiner on the first examination will be similarly diagnosed by the same examiner on the second examination, or the probability that a tooth or surface diagnosed as unhealthy by the examiner on the first examination will be similarly diagnosed on the second examination.

Clinical Study

All data were tested for Normality by plotting in ascending order against the corresponding Normal scores. A normal distribution was indicated by a reasonably straight line plot with no marked concavity or convexity. All data proved to be normally distributed. Paired t-tests were utilised for each treatment cell to determine if differences between readings at baseline and at scheduled examination times were statistically significant at the 95% confidence level. Similarly, at each time point, any differences between the dentifrices and their effects on plaque and gingival scores were tested for statistical significance by means of a two sample t-test.

Confidence intervals were also calculated, and only probabilities of less than or equal to 0.05 were considered to indicate a significant difference between means. To avoid bias, all plaque and gingival scores were weighted for each individual to give the total as derived from either 28 teeth or 168 units as appropriate.

4.2. Results

4.2.1. Reproducibility Study

Plaque accumulation

The unweighted kappa values for plaque presence/absence and plaque severity were 0.61 and 0.60 respectively. Weighted kappa for plaque severity was 0.56 indicating reasonable agreement between the two sets of examination data. For the recording of plaque severity scores (0, 1, 2, 3), the most unreliable score was '0' (absence of plaque), $K = 0.37$ as compared with score '1' $K = 0.58$, score '2' $K = 0.82$. A plausible explanation for this observation may be that if plaque was present on the first examination, but inadvertently removed, it would show as absent at the second examination. The converse would also be true, plaque assessed as absent on the first examination and diagnosed as present at the second. Percentage reproducibility for both plaque presence/absence and severity are shown in **Tables 4.1.-4.2..**

Gingival Condition

Unweighted Kappa values for both gingival colour and bleeding (presence/absence) were 0.40 and 0.23 respectively.

Dice's Coincidence Index for colour, Dice (normal) 46%, and Dice (abnormal) 93%, indicated a probability of repeating a normal score and an abnormal score was 46% and 93% respectively.

The weighted Kappa values for gingival severity scores (0, 1, 2, 3) was 0.26 which indicated only fair agreement between the two sets of examination data. These values perhaps underline the difficulties in obtaining good repeatability data from the assessment of gingival

condition, in particular with gingival bleeding from probing. For example, if gingival inflammation is diagnosed as present on the first examination, but with the absence of bleeding, it is scored as 1. If bleeding is present, however, depending on its severity, it is scored 2 or 3.

The difficulty arises when on the first examination, no bleeding occurs, but when probed on the second occasion bleeding is observed and vice versa. Percentage reproducibility for gingival colour and bleeding are shown in **Tables 4.3.-4.4..**

4.2.2. Clinical Study

Plaque accumulation

There was a slight increase in plaque accumulation in the first two weeks from baseline (**Table 4.5., Fig. 4.3.**), but relatively negligible change thereafter. The effect was identical in both groups. Paired t-tests demonstrated that any changes in the mean scores over the 8-week period were negligible in terms of the total possible score variation, and that there was no evidence that any apparent change in the mean score reflected an actual change in magnitude. Unpaired t-tests indicated no detectable differences between the groups at any time point.

Further analysis demonstrated that over the 8-week period clinical changes as measured by the prevalence of sites with minimal plaque (0/1) and 2/3 scores were also negligible (**Figs. 4.4.-4.5.**).

Gingival Condition

There was a slight increase in Gingival Index in the first two weeks from baseline (**Table 4.6., Fig. 4.6.**), but relatively negligible change thereafter. Gingival Index (GI) tended to be higher in the control (diatomaceous earth) than in the test (silica-based) group. Paired t-tests demonstrated that any changes in mean GI over the 8-week period were negligible in terms of the total possible scores variation, and

there was no evidence that any apparent change in mean score reflected an actual change in magnitude. Unpaired t-tests indicated no detectable differences between the groups at any time point.

Further analysis of gingival scores demonstrated that over the 8-week study period clinical changes, as measured by the prevalence of sites with 0/1 and 2/3 scores, were also negligible (**Figs. 4.7.-4.8.**).

Table 4.1.Plaque Absence or Presence % Reproducibility (324 sites)

No plaque (-1)	Exact Reproducibility (0)	Plaque present (+1)
12	286	26

% Reproducibility 88.3%

Table 4.2.Plaque Severity % Reproducibility (324 sites)

No Plaque (-2) (-1)	Exact Reproducibility (0)	Plaque present (+1) (+2)
0 21	271	31 1

% Reproducibility 83.6%

Table 4.3.Gingival Severity (Bleeding) % Reproducibility (324 sites)

No Bleeding (-3) (-2) (-1)			Exact Reproducibility (0)	Bleeding +1 +2 +3		
0	7	35	198	77	7	0

% Reproducibility 61.1%

Table 4.4.Gingival Severity (colour) % Reproducibility (324 sites)

Colour Change (-2) (-1)		Exact Reproducibility (0)	Colour Change (+1) (+2)	
6	9	288	14	7

% Reproducibility 88.9%

Table 4.5.

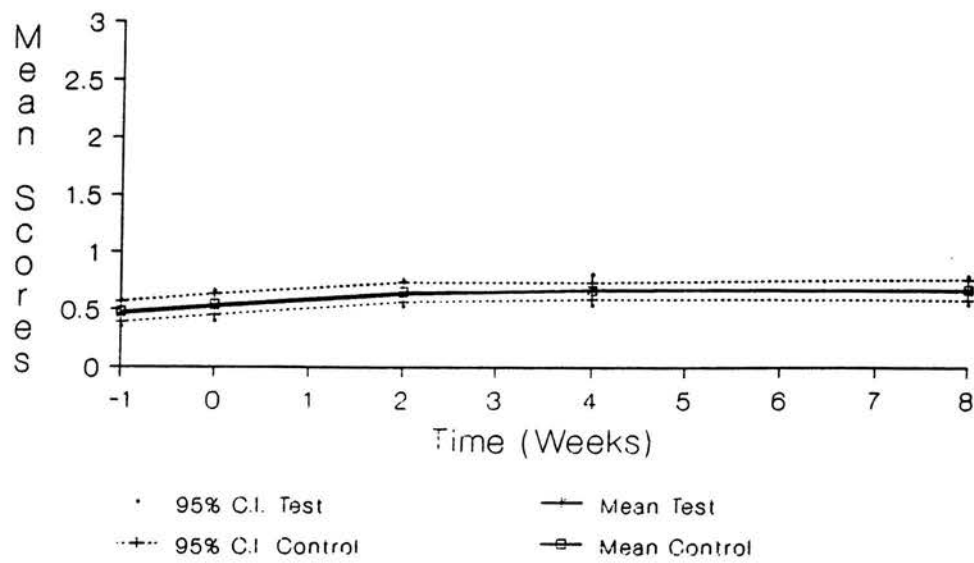
Comparison of mean Plaque Index between test and control groups during the 8 week study

Time	Gp	N	Mean	SD	SEM
Baseline	Test	20	0.50	0.238	0.053
	Control	20	0.51	0.183	0.040
2-wk	Test	20	0.64	0.241	0.054
	Control	20	0.65	0.179	0.040
4-wk	Test	20	0.67	0.287	0.064
	Control	20	0.66	0.158	0.035
8-wk	Test	20	0.67	0.257	0.058
	Control	20	0.68	0.195	0.044
B-2 wk	Test	20	-0.14	0.186	0.042
	Control	20	-0.14	0.162	0.036
B-4 wk	Test	20	-0.18	0.169	0.038
	Control	20	-0.15	0.174	0.039
B-8 wk	Test	20	-0.17	0.158	0.035
	Control	20	-0.17	0.223	0.050

Test = Silica-based group

Control = Diatomaceous earth group

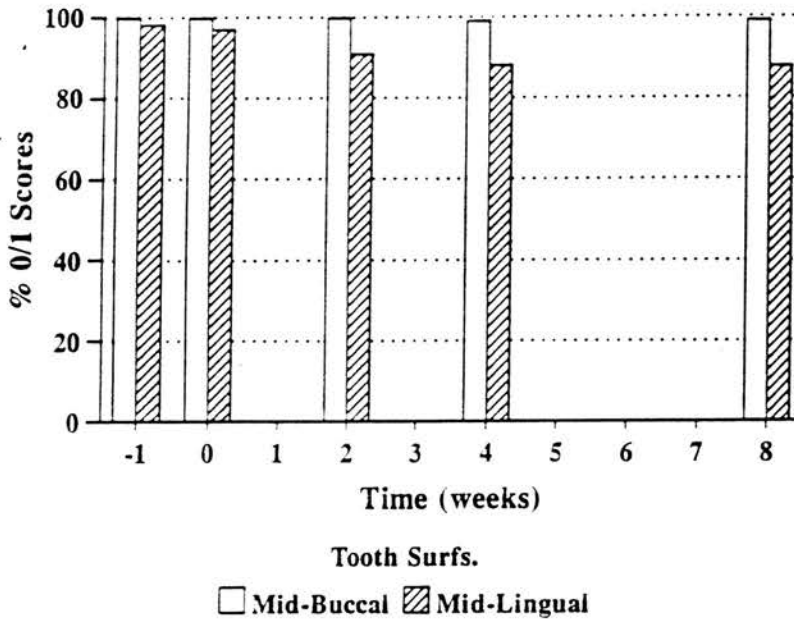
Figure 4.3.



Test = Silica-based group
Control = Diatomaceous earth group

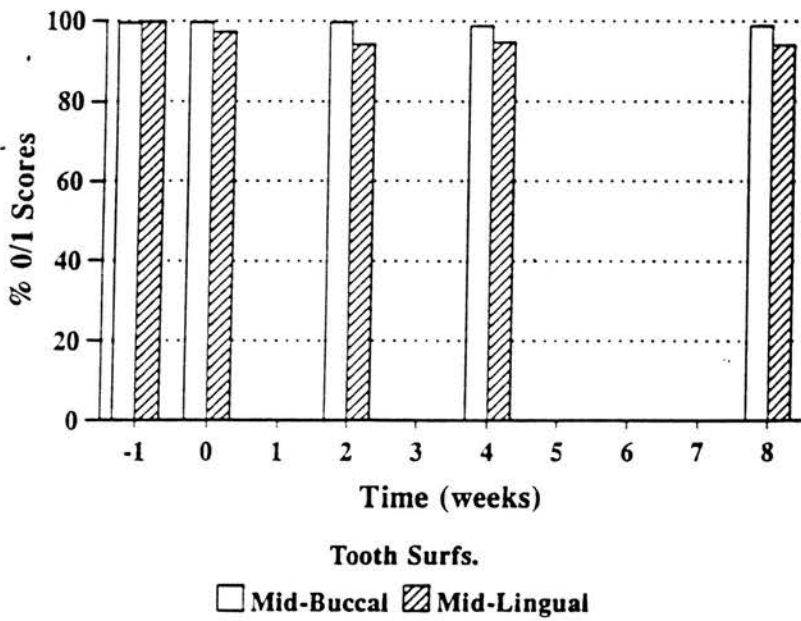
Comparison of mean Plaque Index scores between test and control groups during the 8 week study (Including 95% Confidence Intervals)

Figure 4.4.(a)



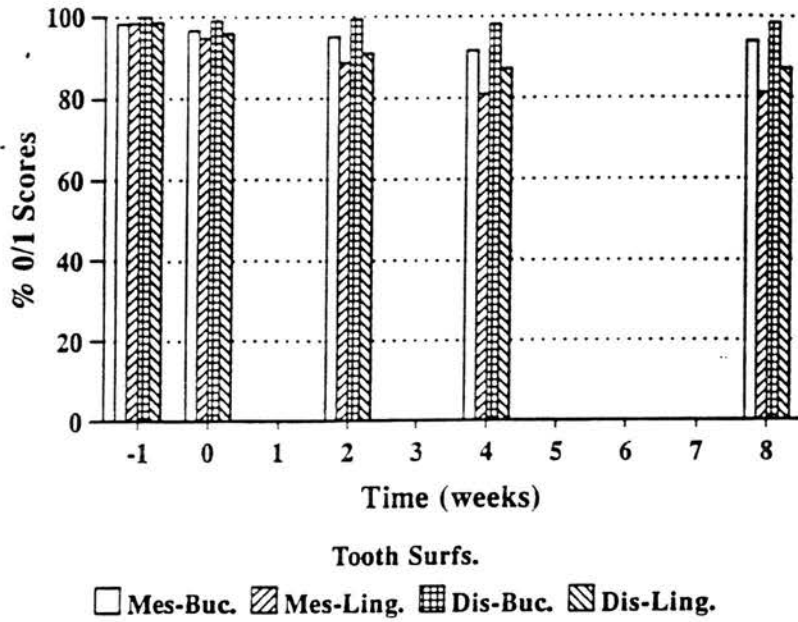
Mean Percentage 0/1 Plaque Index scores for mid-buccal and mid-lingual sites (Silica-based group)

Figure 4.4.(b)



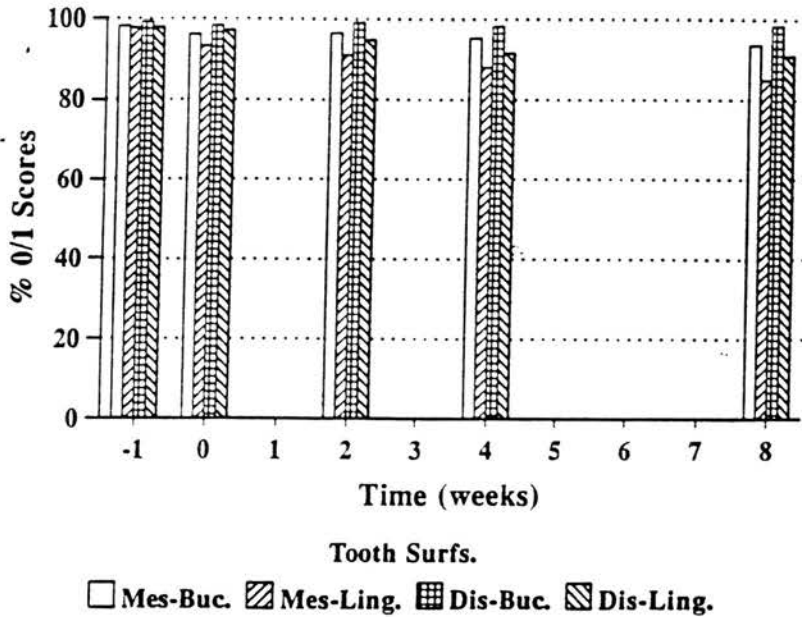
Mean Percentage 0/1 Plaque Index scores for mid-buccal and mid-lingual sites (Diatomaceous earth group)

Figure 4.5.(a)



Mean Percentage 0/1 Plaque Index scores for mesio-buccal, mesio-lingual, disto-buccal and disto-lingual sites (Silica-based group)

Figure 4.5.(b)



Mean Percentage 0/1 Plaque Index scores for mesio-buccal, mesio-lingual, disto-buccal and disto-lingual sites (Diatomaceous earth group)

Table 4.6.

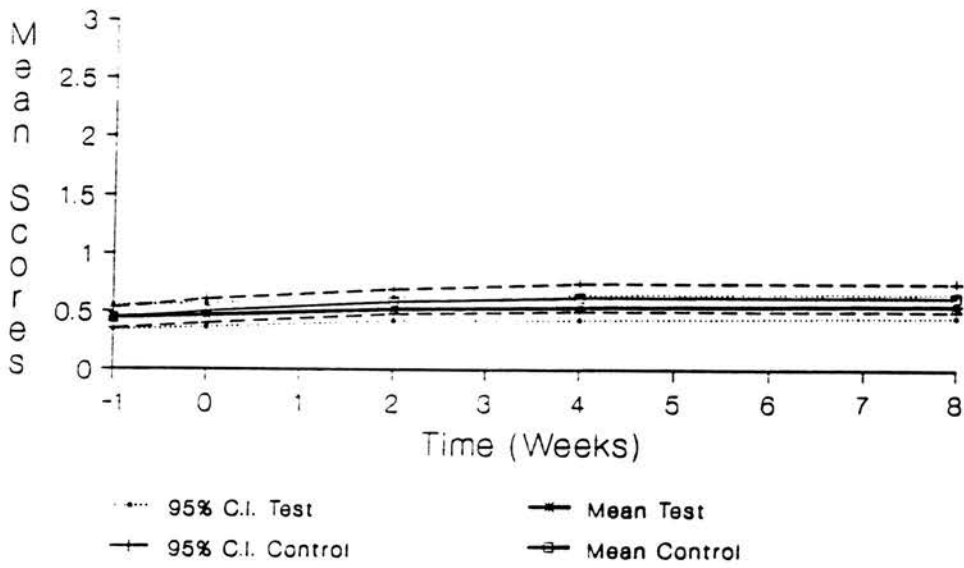
Comparison of mean Gingival Index between test and control groups during the 8 week study

Time	Gp	N	Mean	SD	SEM
Baseline	Test	20	0.46	0.216	0.048
	Control	20	0.47	0.190	0.043
2-wk	Test	20	0.52	0.216	0.048
	Control	20	0.59	0.229	0.051
4-wk	Test	20	0.54	0.236	0.053
	Control	20	0.62	0.260	0.058
8-wk	Test	20	0.56	0.221	0.050
	Control	20	0.63	0.263	0.059
B-2 wk	Test	20	-0.07	0.163	0.037
	Control	20	-0.11	0.139	0.031
B-4 wk	Test	20	-0.08	0.173	0.039
	Control	20	-0.15	0.156	0.035
B-8 wk	Test	20	-0.16	0.199	0.045
	Control	20	-0.16	0.160	0.035

Test = Silica-based group

Control = Diatomaceous earth group

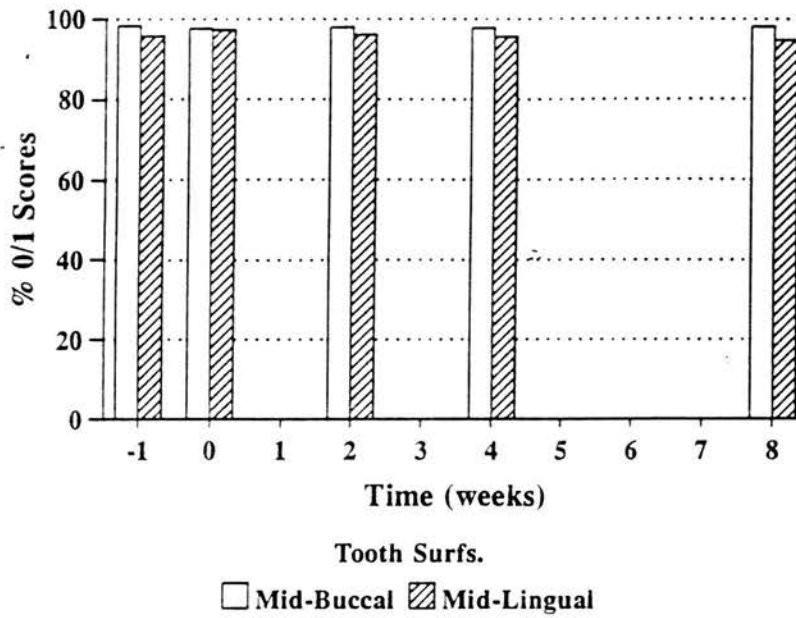
Figure 4.6.



Test = Silica-based group
 Control = Diatomaceous earth group

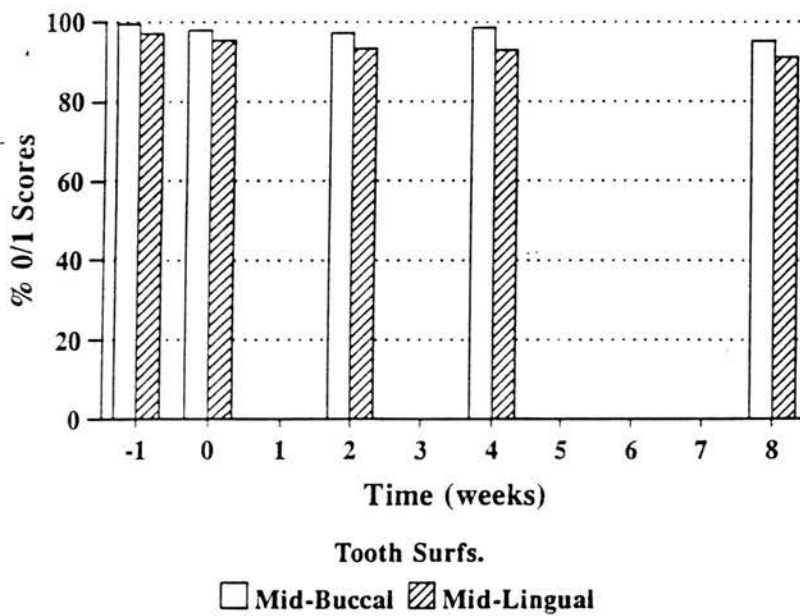
Comparison of mean Gingival Index scores between test and control groups during the 8 week study (Including 95% Confidence Intervals)

Figure 4.7.(a)



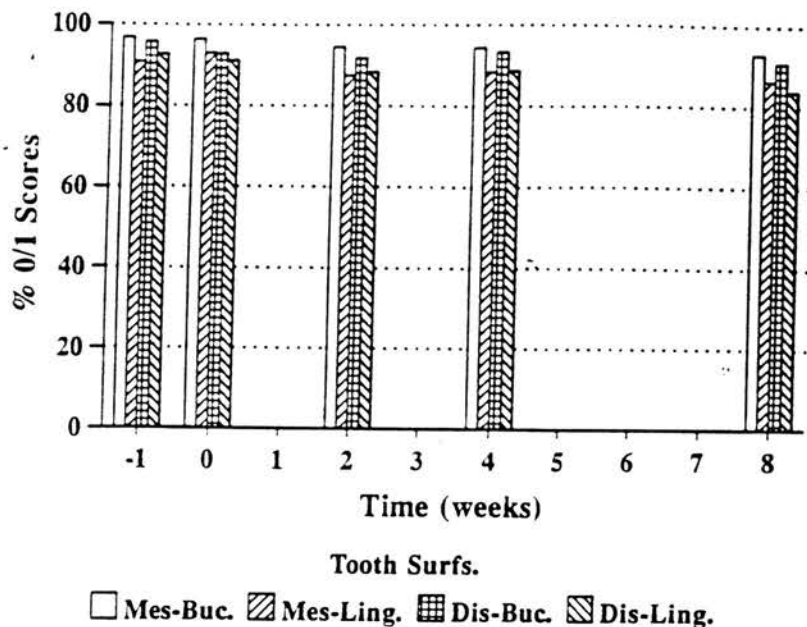
Mean Percentage 0/1 Gingival Index scores for mid-buccal and mid-lingual sites (Silica-based group)

Figure 4.7.(b)



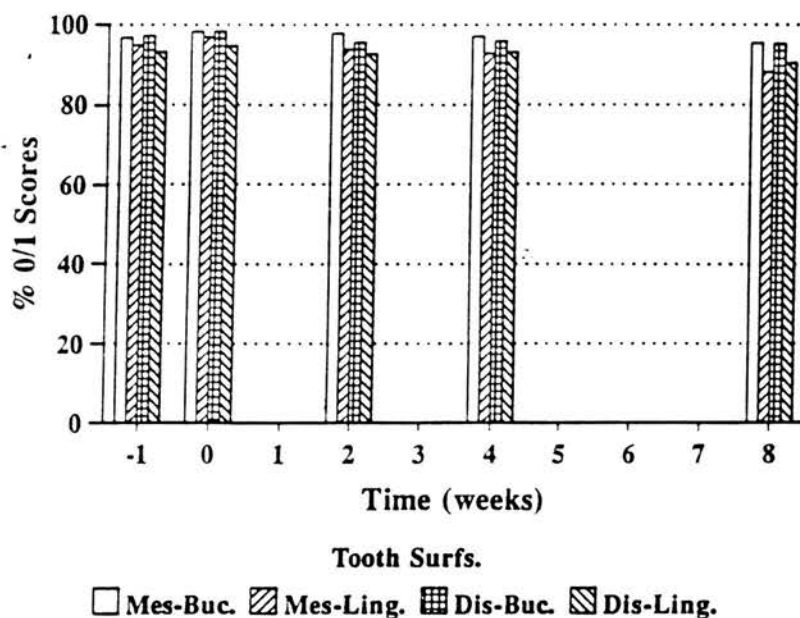
Mean Percentage 0/1 Gingival Index scores for mid-buccal and mid-lingual sites (Diatomaceous earth group)

Figure 4.8. (a)



Mean Percentage 0/1 Gingival Index scores for mesio-buccal, mesio-lingual, disto-buccal and disto-lingual sites (Silica-based group)

Figure 4.8. (b)



Mean Percentage 0/1 Gingival Index scores for mesio-buccal, mesio-lingual, disto-buccal and disto-lingual sites (Diatomaceous earth group)

Discussion

According to Addy et al. 1990b, several investigators have claimed beneficial treatment effects of various desensitizing dentifrices based on the purely subjective evaluation of improved oral hygiene without actually recording the differences in plaque scores between the dentifrices. For example, Toto et al. (1958) reported that the participants' oral hygiene ranged from poor to good, whereas Manochehr-Pour et al. (1984) reported that most of the participants showed an improvement in the course of the study but did not attempt to record plaque. Other investigators (Clark et al. 1985, Silverman 1986, Hovgaard et al. 1988, Salvato et al. 1989, Addy et al. 1990b) attempted to measure plaque by partial or whole mouth recording utilising the Greene & Vermillion (1960) or Silness & L  e (1964) indices.

Several desensitizing dentifrice studies (Zinner et al. 1977, Gedalia et al. 1978, Silverman 1985, Addy et al. 1990) made no attempt to change the oral hygiene practices of participants during the course of the study, whereas Shapiro et al. (1970) and Hovgaard et al. (1988) attempted to carefully control oral hygiene procedures by instruction, reinforced at each visits and corrected if required (Shapiro et al. 1970). Other investigators (Gedalia et al. 1978, Clark et al. 1985), however, found that even when oral hygiene procedures were not changed prior to inclusion in desensitization dentifrice studies, that there was little significant difference in plaque index between the groups.

In the present study, no attempt was made to change the participants' oral hygiene, but all patients received oral hygiene instruction and debridement prior to inclusion of the study, which may account for the relatively low plaque and gingival index scores at the commencement of the study. The slight increase in plaque and gingival scores in the two weeks following baseline readings, and the levelling out of the mean values, may be explained by a slight lapse following prebaseline treatment and subsequent stabilised maintenance thereafter (Garcia-Godoy et al. 1992). It was also observed that no further change in PlI

and GI took place after two weeks. There was no evidence to suggest that any apparent change in the mean plaque and gingival scores reflected an actual change in magnitude. Neither plaque accumulation nor gingival condition significantly changed from baseline levels during the course of the study. The results of the present study appear to confirm the observations of Gedalia et al. (1978) and Clark et al. (1985) in that there was little or no change between the two groups in plaque scores. Indeed the plaque effect was identical in both test and control groups. In conclusion, there was no evidence to suggest that SCH dentifrices increased plaque accumulation, or that the abrasivity of the desensitizing dentifrice affected the level of plaque.

The results of this study, therefore, do not support the conclusions of previous studies which indicated that SCH dentifrices increased plaque accumulation. It was notable that neither SCH dentifrices had any clinical effect per se on plaque or gingival condition.

CHAPTER 5

QUANTIFICATION OF PAIN IN CERVICAL DENTINAL SENSITIVITY (CDS) STUDIES - Subjective response

Introduction

Traditionally CDS has been evaluated mainly subjectively on the basis of the individual patient's subjective evaluation of the level of pain elicited by mechanical, thermal, electrical and chemical stimuli. The method and interpretation of pain assessment from these stimuli, however, is open to question and interpretation (**section 1.3.**). Furthermore, the subjective nature of the response may also complicate assessment of pain arising from CDS.

Therefore, two studies were undertaken to run current with the 8-week CDS study described in Chapter 2.

The purpose of the first study was to compare four methods of assessment of the pain associated with CDS (continuous Visual Analogue Scale [VAS], 0-10 Numerical Rating VAS scale [NRS], and separate intensity verbal descriptor [IVD] and unpleasantness verbal descriptor [UVD] word scales) following tactile and thermal stimulation; together with an overall assessment of perception to daily stimuli in patients presenting with cervical dentinal sensitivity (CDS). The overall aim was to establish the usefulness and comparability of each of the described methods in the assessment of such pain.

The second study involved 40 patients from the 8-week study who were asked to indicate which word descriptors from a McGill Pain Questionnaire (MPQ) best described their perception of pain arising from CDS.

This was completed on two occasions (0 and 56 days) and the selected word choices analysed (**section 5.3.2.**).

5.1. Materials & Methods

5.1.1. Comparison of methods of subjective evaluation

Prior to tactile and thermal stimulation

Patients were asked to rate their perception of sensitivity to hot/cold food and drink, to toothbrushing, and to sweet and sour food by:

- 1). Placing a mark on a 10cm line (continuous VAS). The distance of the mark from the "no pain" end provided an estimate of pain perceived by the patient and constituted an Overall Sensitivity, Tactile, and Subjective Air score respectively (**Fig. 5.1.**).
- 2). Placing a mark on a 0-10 NRS form (**Fig. 5.2.**).
- 3). Identifying intensity or unpleasantness words from the verbal descriptor list which most closely resembled their own perception of discomfort (**Tables 5.1.-5.2.**).

Tactile stimulation (Yeaple Probe)

Patients were asked to rate their perception of sensitivity experienced following Yeaple probe application to the exposed cervical area of the root dentine in the manner described above.

Thermal stimulation (cold air: dental unit syringe)

Ten minutes after tactile stimulation, patient response was assessed to a one-second application of cold air (dental air syringe) at a temperature of 19°C-24°C, 40-65 p.s.i..

5.1.2. McGill Pain Questionnaire study

40 patients from the original 8-week study were shown 20 sets of words from a MPQ form (**Table 5.3.**) and asked to select a word from each set which best described their present pain or discomfort arising from CDS.

Figure 5.1.

VISUAL ANALOGUE SCALE (VAS) cm

CONTINUOUS VAS (0-10)

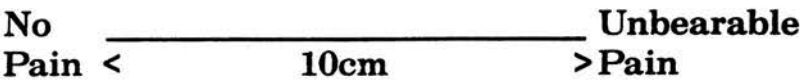


Figure 5.2.

NUMERICAL RATING SCALE (NRS)

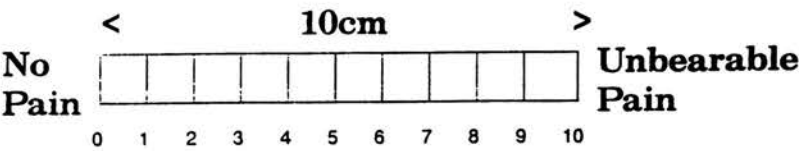


Table 5.1.INTENSITY WORD DESCRIPTORS

0	NO SENSATION
1	BARELY PERCEPTIBLE
2	VERY MILD
3	MILD
4	MODERATE
5	BARELY STRONG
6	STRONG
7	INTENSE
8	VERY INTENSE
9	EXTREMELY INTENSE
10	THE MOST INTENSE SENSATION IMAGINABLE

Table 5.2.UNPLEASANTNESS WORD DESCRIPTORS

0	NOTHING
1	NOT BAD AT ALL
2	ANNOYING
3	UNPLEASANT
4	DISAGREEABLE
5	SLIGHTLY DISTRESSING
6	DISTRESSING
7	INTOLERABLE
8	THE MOST UNPLEASANT IMAGINABLE

N.B. Numbers were not shown to patients

5.1.3. Data Analysis

Since the data obtained from the first study were derived from highly subjective assessments, an analysis of the overall trend of the results rather than detailed statistical analysis of isolated scores was indicated. To this end, a moving average method was used in order to smooth out the otherwise inevitably erratic high and low points; for example if pain is rated by a subject at level 3, this is almost certainly indicating not so much an exact level 3 stimulus (which in any case is not possible to define), but rather pain within the general area arbitrarily delineated by a measure of 3. To allow for this imprecision, in preparing Figures 5.4, 5.6, and 5.8 the value recorded at, say, the '3' point on the baseline scale is obtained by calculating the unweighted mean of the total reported '2', '3', and '4' ratings. Similarly, the '4' point on the scale is obtained by taking the unweighted mean of the total reported '3', '4' and '5' ratings (**Figs. 5.3.-5.8.**).

For the second study each patient's contribution to the total number of scores were weighted. A Wilcoxon two-sample rank test was used to examine the changes in score between the two groups.

5.2. Results

5.2.1. Comparison of methods of subjective evaluation

Twenty five patients (8M + 17F) mean age 42.6 years (SD 9.7) who provided voluntary written informed consent participated in the study.

- 1). For overall sensitivity scores, 0-10 NRS, IVD and UVD assessment appeared to provide reasonable alternatives to continuous VAS (**Figs. 5.3.-5.4.**).
- 2). Overall, both 0-10 NRS and IVD scores appeared to provide reasonable alternatives to continuous VAS assessment (**Figs. 5.3.-5.8.**).
- 3). The unpleasantness scale (UVD) appeared to be of little or no

Table 5.3.

SENSITIVITY DESCRIPTORS

Which words best describe the present pain of your sensitive teeth. Circle a single word in each group that best describes the pain. Leave out any word-group that is not appropriate to the current pain of your sensitive teeth.

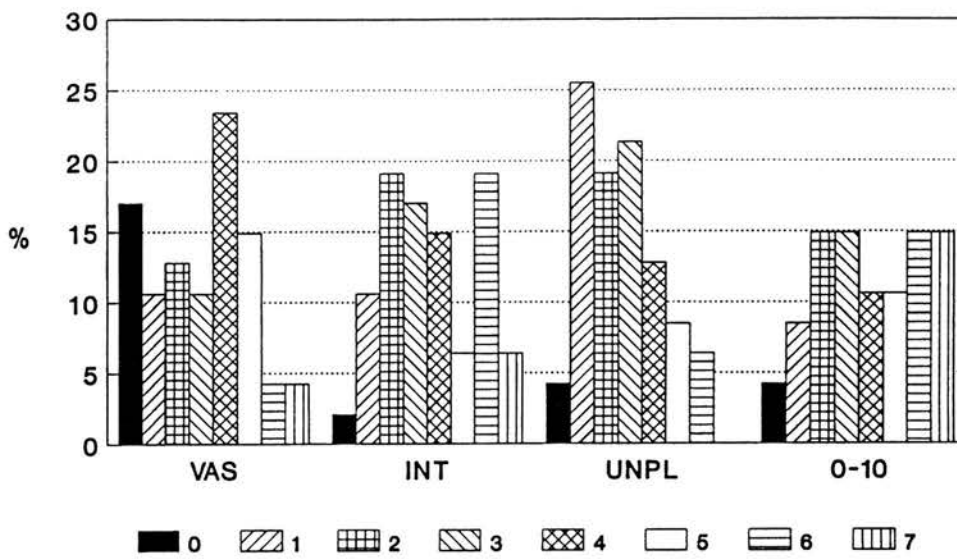
1	2	3	4
1. Flickering	1. Jumping	1. Pricking	1. Sharp
2. Quivering	2. Flashing	2. Boring	2. Cutting
3. Pulsing	3. Shooting	3. Drilling	3. Lacerating
4. Throbbing		4. Stabbing	
5. Beating		5. Lancinating	
6. Pounding			
5	6	7	8
1. Pinching	1. Tugging	1. Hot	1. Tingling
2. Pressing	2. Pulling	2. Burning	2. Itching
3. Gnawing	3. Wrenching	3. Scalding	3. Smarting
4. Cramping		4. Searing	4. Stinging
9	10	11	12
1. Dull	1. Tender	1. Tiring	1. Sickening
2. Sore	2. Taut	2. Exhausting	2. Suffocating
3. Hurting	3. Rasping		
4. Aching	4. Splitting		
5. Heavy			
13	14	15	16
1. Fearful	1. Punishing	1. Wretched	1. Annoying
2. Frightful	2. Gruelling	2. Blinding	2. Troublesome
3. Terrifying	3. Cruel		3. Miserable
	4. Vicious		4. Intense
	5. Killing		5. Unbearable
17	18	19	20
1. Spreading	1. Tight	1. Cool	1. Nagging
2. Radiating	2. Numb	2. Cold	2. Nauseating
3. Penetrating	3. Drawing	3. Freezing	3. Agonizing
4. Piercing	4. Squeezing		4. Dreadful
	5. Tearing		5. Torturing

Word Descriptors (McGill Pain Questionnaire MPQ)

value in its present form, as it did not provide accuracy or sensitivity when assessing pain arising from CDS, except at very low levels of discomfort (**Figs. 5.3.-5.8.**).

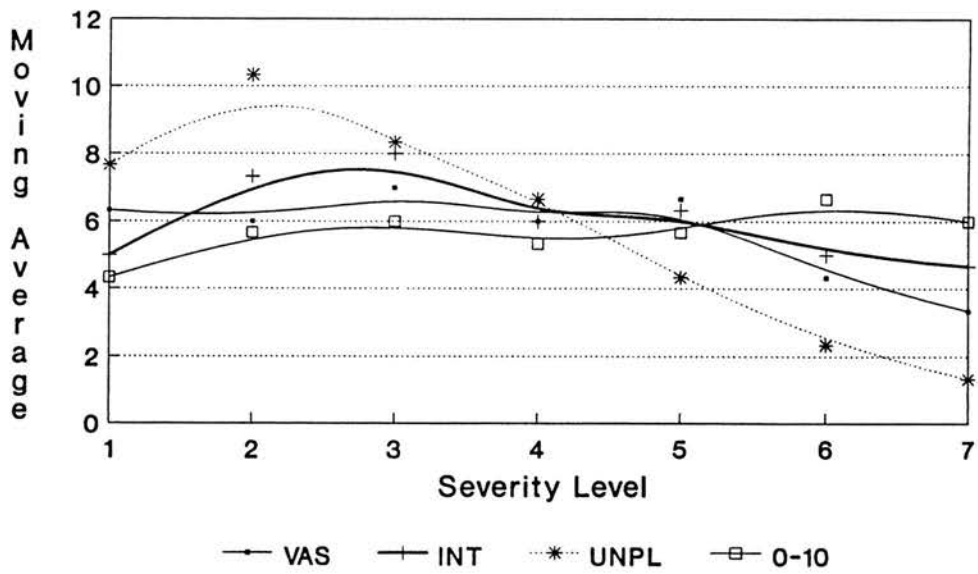
- 4). Comparison of all four methods demonstrated that tactile stimulation (Yeaple probe) caused the least discomfort (**Figs. 5.5-5.6.**).
- 5). For tactile sensitivity, both continuous VAS and 0-10 NRS scores were in good agreement, as were intensity (IVD) and unpleasantness (UVD) descriptor words (**Figs.5.5.-5.6.**).
- 6). All four methods demonstrated that air stimulation (dental unit syringe) was perceived by patients to cause the greater discomfort (**Figs. 5.7.-5.8.**).
- 7). For air sensitivity, continuous VAS peaked at 2-4, while IVD and 0-10 NRS peaked at 3-5 and 3-6 respectively. Unpleasantness (UVD) scores peaked at two levels (2-4 & 6), which would appear to indicate that the choice of word is important and that the descriptor word (score 5), "slightly distressing" needs replacing, while the descriptor word (score 6), "distressing" is a better descriptor of score 3-4 (**Figs. 5.7.-5.8.**).

Figure 5.3.



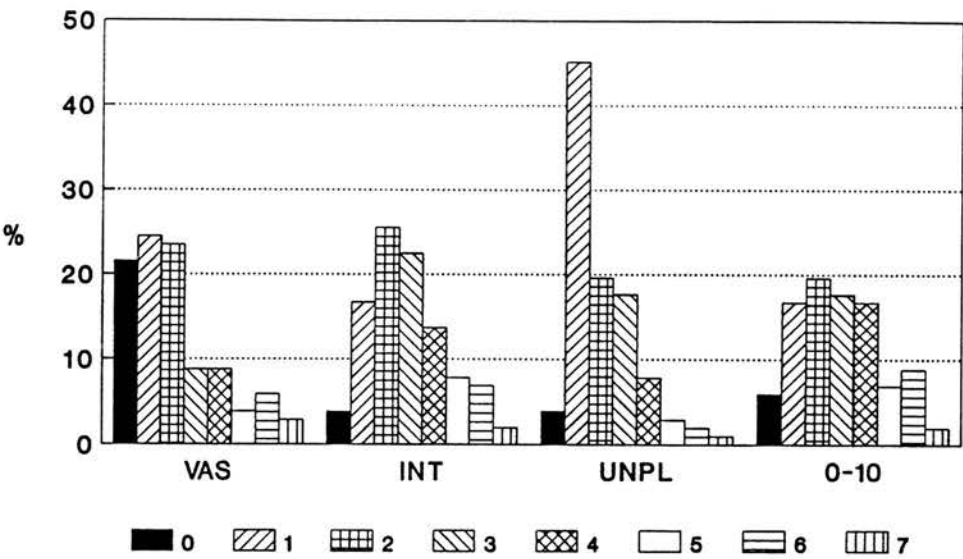
Mean Overall Sensitivity scores

Figure 5.4.



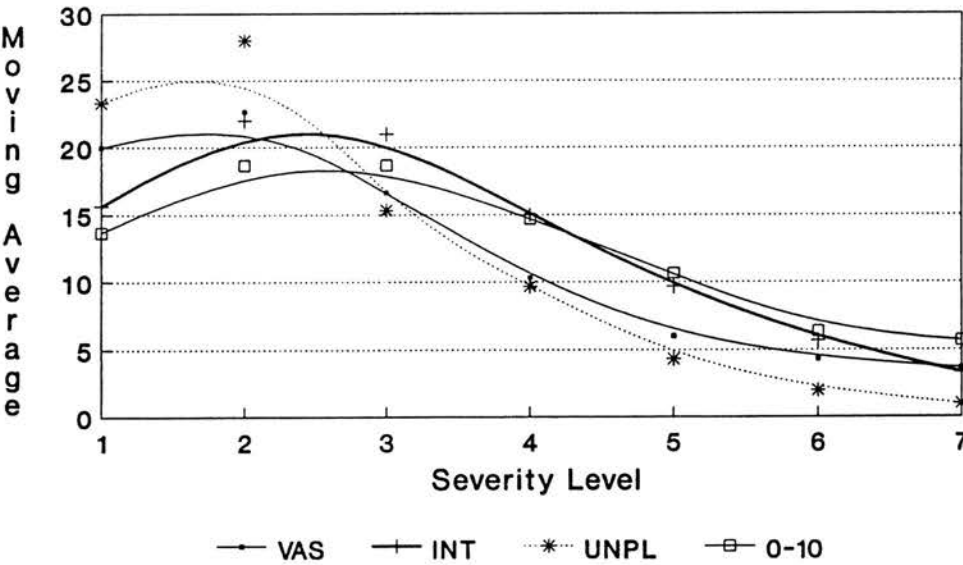
Overall Sensitivity scores (Moving average analysis)

Figure 5.5.



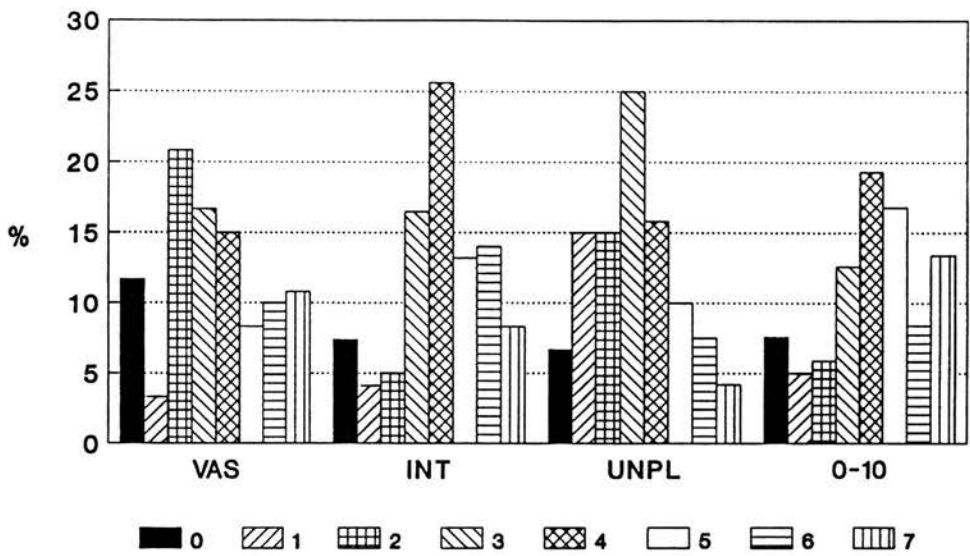
Mean Tactile sensitivity scores (Yeaple probe)

Figure 5.6.



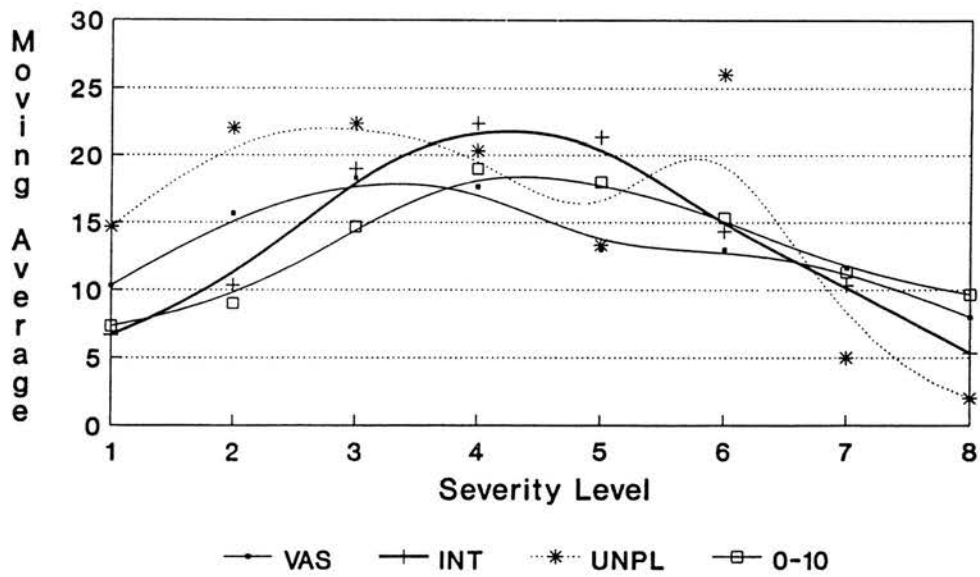
Tactile sensitivity scores (Moving average analysis)

Figure 5.7.



Mean thermal stimulus response scores (cold air blast)

Figure 5.8.



Thermal stimulus response scores (Moving average analysis)

5.2.2. McGill Pain Questionnaire study

All 40 patients from the 8-week clinical study completed a MPQ form at 0 and 56 days.

The raw data for both groups (A & B) can be observed in **Tables 5.4.-5.5..** Consistent returns for each word group are in bold type.

A summary of the totals showing a) the percentage of consistent pairings; b) the percentage of responses where a lowered numbered word was given on the second visit; and c) the percentage where a higher numbered word was given on the second visit (**Table 5.6.**).

A SND test of the difference in proportions showing a higher score on the second visit between the two groups was not significant (SND = 1.39 [unweighted]; 0.59 [weighted]).

A Wilcoxon two-sample rank test was used to look at the changes in score between the two groups. Thus if a score of '2' was given on the first visit, but '1' on the second, this is scored as a change of '+1'. If the score was '1' on the first visit, '2' on the second, a change of '-1' would be recorded. If the scores were recorded as '1' at both visits, then there would be a change of '0'. The test was carried out on all 130 scores in Gp A and 113 scores in Gp B regardless of subject. The result was not significant (SND = 1.26).

Each patient's contribution to the total number of scores was weighted. The average number of scores for Gp A was 6.5 and for Gp B 5.65 (Average score for both groups 6.1). The contribution of each patient was, therefore weighted as if they had been contributing that number of scores. For example, if a patient contributed 19 scores of which 9 were 'consistent', this is the equivalent of $9 \times 6.5/19 = 3.08$, if the total of scores had been 6.5 instead of 19.

The number of times each word in each word set was most frequently mentioned, together with the overall popularity of each word quoted, is listed in **Tables 5.7.-5.8., Fig. 5.9..**

Table 5.4.MPQ Word Descriptor choices (Gp A)

Pt No↓	1→	2	3	4	5	6	7	8	9	10
5	-	2	2	1	3*	2	2	3	5	1
	5	1	4	2	3*	2	2	2	2	3
7	-	-	-	-	-	-	-	-	-	-
	2	-	-	-	-	-	-	-	-	-
8	4	3	-	-	-	-	-	-	4	-
	4	-	-	-	3	-	-	-	4	1
9	4	1	-	-	-	1	-	-	-	-
	-	-	5	3	-	-	-	-	-	-
11	-	3	4	1	-	-	4	1	-	-
	1	2	4	1	-	-	2	1	-	-
12	-	3	-	1	-	-	-	-	-	-
	1	-	-	-	-	-	-	1	-	-
13	-	1	2	1	2	2	1	4	2	1
	1	3	2	1	4	-	4	4	3	3
14	-	-	1	-	-	-	-	1	4	1
	1	-	1	-	1	-	-	1	1	1
17	3	2	4	1	-	-	2	2	4	3
	3	1	-	-	-	-	3	2	-	-
19	-	-	-	1	-	-	-	-	-	-
	-	3	4	1	-	-	-	4	-	-

Group A = Silica-based group

Key to Tables 5.4.-5.5.

↓ = Pt No

→ = Word group No (1-20)

* Matching pairs in bold type

Table 5.4. (cont.)MPQ Word Descriptor choices (Gp A)

Pt No	11	12	13	14	15	16	17	18	19	20
5	1	2	3	1	1	3	2	3	1	4
	1	2	1	4	1	3	1	3	2	4
7	-	-	-	-	-	-	-	-	3	-
	-	-	-	-	-	-	-	-	3	-
8	-	-	-	-	-	2	2	-	-	-
	1	-	-	-	-	1	-	-	-	-
9	-	-	-	5	-	-	-	-	-	3
	-	-	-	-	-	4	-	-	-	3
11	-	-	-	-	-	1	4	-	3	-
	-	-	-	-	-	1	4	-	-	1
12	-	-	-	-	-	-	-	-	-	-
	-	-	-	-	-	1	-	-	-	-
13	1	1	2	5	2	1	2	5	1	4
	2	1	3	5	2	1	-	5	1	4
14	-	-	-	-	-	2	-	-	-	1
	-	-	-	-	-	2	-	-	-	1
17	-	-	2	-	-	1	1	-	3	1
	-	-	-	-	-	1	-	-	-	1
19	-	-	-	-	-	5	-	-	-	-
	-	-	-	-	-	1	2	-	-	-

Gp A = Silica-based group

Table 5.4. (cont.)MPQ Word Descriptor choices (Gp A)

Pt No	1	2	3	4	5	6	7	8	9	10
22	-	3	4	-	-	-	-	-	-	-
	-	3	4	1	-	-	-	3	-	-
23	3	3	1	1	2	-	-	1	3	1
	-	2	2	1	2	1	-	2	4	1
24	4	3	4	1	-	-	-	-	4	1
	-	-	-	1	3	-	-	-	4	1
26	4	-	-	-	-	-	-	-	-	1
	4	3	-	-	-	-	-	-	4	1
29	-	3	-	1	-	-	-	2	2	-
	-	3	-	1	-	-	-	-	-	-
30	-	3	3	1	1	2	1	3	1	1
	1	1	4	1	2	2	4	2	1	1
32	-	-	-	1	-	-	4	-	4	-
	4	3	4	1	-	-	-	-	4	-
37	-	3	4	1	-	-	-	4	3	1
	-	2	4	1	-	-	4	4	4	-
38	2	3	3	1	-	-	4	-	-	1
	2	3	3	-	-	-	-	1	-	1
40	-	3	-	-	-	-	1	3	4	1
	4	3	-	-	-	-	-	-	2	1

Gp A = Silica-based group

Table 5.4. (cont.)MPQ Word Descriptor choices (Gp A)

Pt No	11	12	13	14	15	16	17	18	19	20
22	-	-	-	-	-	1	-	-	2	-
	-	-	-	-	-	1	-	-	-	-
23	-	-	-	-	-	2	4	1	2	1
	1	1	1	-	-	1	3	1	1	1
24	-	-	-	-	-	2	3	-	-	1
	-	-	1	-	-	3	3	-	-	1
26	-	-	-	-	-	1	-	2	-	-
	-	-	-	-	-	-	-	3	-	-
29	-	-	-	-	-	-	-	-	-	-
	-	-	-	-	-	-	-	-	2	-
30	2	1	3	1	1	3	1	2	2	4
	2	2	1	1	1	4	1	2	2	1
32	-	-	-	-	-	4	1	-	-	1
	-	-	-	-	2	2	4	-	3	3
37	-	-	-	-	-	4	4	-	-	4
	-	-	-	-	-	4	4	-	-	4
38	-	1	-	-	-	4	3	3	3	3
	-	-	-	-	-	5	2	-	2	-
40	-	-	-	-	-	-	-	-	-	-
	-	-	-	-	-	2	-	-	-	-

Gp A = Silica-based group

Table 5.5.MPQ Word Descriptor choices (Gp B)

Pt No	1	2	3	4	5	6	7	8	9	10
1	-	3	4	1	-	-	-	-	-	1
	-	-	-	1	-	-	-	-	-	1
2	-	1	4	1	-	-	-	-	-	-
	-	3	4	1	-	-	-	-	4	-
3	4	2	-	-	-	-	2	4	2	1
	3	1	4	1	3	2	2	3	2	1
4	3	3	4	-	-	-	4	-	1	-
	1	3	-	-	-	-	-	1	1	1
6	-	3	-	1	-	-	-	-	4	1
	-	1	4	2	-	-	-	-	3	-
10	-	2	-	4	-	-	-	3	-	1
	4	2	4	1	-	-	4	3	3	3
15	-	3	-	1	-	-	-	-	3	1
	-	3	2	1	-	-	-	4	-	2
16	4	-	-	-	2	-	-	-	4	-
	3	3	4	1	2	3	4	4	4	1
18	-	3	-	-	-	-	-	1	2	1
	-	3	4	-	-	-	-	-	-	-
20	4	3	-	1	-	-	4	-	3	1
	4	3	-	-	-	-	-	-	2	1

Gp B = Diatomaceous earth group

Table 5.5.(cont.)MPQ Word Descriptor choices (Gp B)

Pt No	1	2	3	4	5	6	7	8	9	10
21	-	2	1	1	-	-	1	1	2	1
	2	-	1	1	-	-	-	4	3	1
25	-	3	3	1	4	-	-	4	2	1
	5	-	-	-	1	2	-	1	3	-
27	4	3	-	-	-	-	-	-	4	-
	4	3	-	-	-	-	-	-	1	-
28	4	3	4	1	-	-	-	-	4	1
	3	2	4	1	-	-	1	4	4	1
31	1	3	1	1	3	3	2	4	4	1
	4	3	4	1	3	1	2	1	3	1
33	4	-	-	-	-	-	-	-	-	1
	-	-	-	-	-	-	-	1	-	-
34	-	-	4	1	-	-	-	-	4	-
	-	-	-	1	3	-	-	-	4	-
35	6	-	-	1	-	-	-	1	4	-
	-	-	-	1	-	-	-	1	3	1
36	4	3	-	-	-	-	-	-	4	-
	4	-	-	-	-	-	-	-	-	-
39	-	2	-	-	-	-	-	-	-	-
	-	2	-	-	-	-	-	-	-	-

Gp B = Diatomaceous earth group

Table 5.5.(cont.)MPQ Word Descriptor choices (Gp B)

Pt No	11	12	13	14	15	16	17	18	19	20
1	-	-	-	-	-	-	3	-	-	-
	-	-	-	-	-	1	-	-	-	-
2	-	-	-	-	-	1	3	-	-	-
	-	-	-	-	-	2	4	-	-	-
3	-	-	-	-	-	1	-	-	-	1
	1	-	-	-	-	1	-	-	-	1
4	-	-	-	-	-	1	3	-	-	1
	-	-	-	-	-	-	-	-	-	-
6	-	-	1	5	-	5	3	5	-	3
	-	-	-	-	-	5	3	-	-	3
10	-	-	2	4	-	1	4	-	3	-
	-	-	2	4	-	4	4	-	3	1
15	-	-	-	-	-	-	4	-	-	-
	-	-	-	-	-	4	4	4	-	-
16	-	-	-	2	-	1	3	-	3	-
	1	1	1	1	1	1	3	3	2	1
18	-	-	-	-	-	2	4	-	-	-
	-	-	-	-	-	2	-	-	2	-
20	-	-	1	-	-	3	3	-	-	3
	-	-	-	-	-	2	-	-	-	-

Gp B = Diatomaceous earth group

Table 5.5.(cont.)MPQ Word Descriptor choices (Gp B)

Pt No	11	12	13	14	15	16	17	18	19	20
21	-	-	-	-	-	1	-	-	2	1
	-	-	-	-	-	1	3	-	-	1
25	-	-	3	-	-	1	3	2	3	1
	-	-	1	1	1	2	4	4	2	1
27	-	-	-	-	-	4	4	-	-	-
	-	-	-	-	-	-	4	-	-	-
28	1	-	-	-	1	3	4	-	-	3
	1	1	-	3	2	3	4	-	1	3
31	1	1	1	1	1	2	4	4	2	4
	1	1	1	1	1	2	2	1	2	1
33	-	-	-	-	-	-	4	-	-	-
	-	-	-	-	-	-	-	-	2	-
34	-	-	-	-	-	1	2	-	-	1
	-	-	-	-	-	1	4	-	-	-
35	-	-	-	-	-	5	-	2	2	-
	-	-	-	-	-	-	-	2	2	-
36	-	-	-	-	-	-	-	-	-	-
	-	-	-	-	-	-	-	-	-	-
39	-	-	-	-	-	-	-	-	-	-
	-	-	-	-	-	-	-	-	-	-

Gp B = Diatomaceous earth Gp

Table 5.6.Comparison of raw and weighted data (MPQ)

	Raw	Weighted ¹	Raw	Weighted ²
Same (Consistent)	61%	70%	62%	70%
Higher (2nd visit)	21%	15%	15%	13%
Lower (2nd visit)	18%	14%	23%	18%

¹Group A = Silica-based group²Group B = Diatomaceous earth group

Table 5.7.**Most popular word in each word group (most frequently quoted)**

Gp No	1st choice	2nd choice	3rd choice
1	Throbbing (20)	Flickering/Pulsing (7)	Quivering (4)
2	Shooting (37)	Flashing (12)	Jumping (8)
3	Stabbing (25)	Pricking (6)	Boring (5)
4	Sharp (45)	Cutting (2)	Lacerating (1)
5	Gnawing (8)	Pressing (6)	Pinching (3)
6	Pulling (7)	Tugging (3)	Wrenching (2)
7	Searing (10)	Burning (8)	Hot (5)
8	Tingling (15)	Stinging (12)	Smarting (7)
9	Aching (24)	Hurting (11)	Sore (10)
10	Tender (40)	Rasping (4)	Taut (1)
11	Tiring (11)	Exhausting (3)	---
12	Sickening (9)	Suffocating (3)	---
13	Fearful (10)	Frightful/Terrifying (4)	---
14	Punishing (7)	Killing (4)	Vicious (3)
15	Wretched (9)	Blinding (4)	---
16	Annoying (26)	Troublesome (13)	Intense (8)
17	Piercing (19)	Penetrating (15)	Radiating (7)
18	Numb (6)	Drawing (5)	Tight/Squeezing/ Tearing (3)
19	Cold (16)	Freezing (10)	Cool (5)
20	Nagging (21)	Agonizing (9)	Dreadful (8)

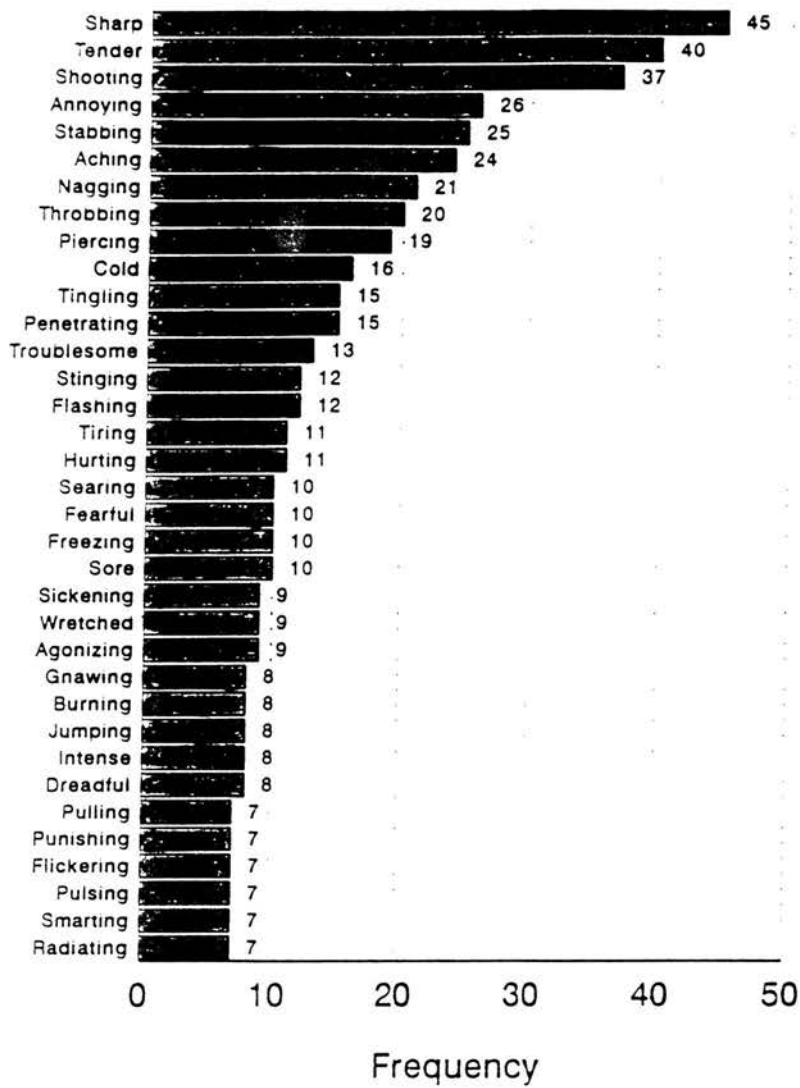
Table 5.8.Overall choice of word descriptor (MPQ)

Descriptor word	Frequency of use	Group Number
Sharp	45	4
Tender	40	10
Shooting	37	2
Annoying	26	16
Stabbing	25	3
Aching	24	9
Nagging	21	20
Throbbing	20	1
Piercing	19	17
Cold	16	19
Tingling	15	8
Penetrating	15	17 (2nd choice)
Troublesome	13	16 (2nd choice)
Stinging	12	8 (2nd choice)
Flashing	12	2 (2nd choice)
Tiring	11	11
Hurting	11	9 (2nd choice)
Searing	10	7
Fearful	10	13

Table 5.8.(cont.)Overall choice of descriptor word (MPQ)

Descriptor Word	Frequency of use	Group Number
Freezing	10	19 (2nd choice)
Sore	10	9 (2nd choice)
Sickening	9	12
Wretched	9	15
Agonizing	9	20 (2nd choice)
Gnawing	8	5
Burning	8	7 (2nd choice)
Jumping	8	8 (2nd choice)
Intense	8	16 (2nd choice)
Dreadful	8	20 (2nd choice)
Pulling	7	6
Punishing	7	14
Flickering	7	1 (2nd choice)
Pulsing	7	1 (2nd choice)
Smarting	7	8 (3rd choice)
Radiating	7	17 (3rd choice)

Figure 5.9.



Overall Choice of Word Descriptor (MPQ)

Discussion

Qualitative evaluation of the subjective response in CDS studies using either verbal and/or non-verbal techniques has been well documented (Brough et al. 1985, Silverman 1985, Clark et al. 1987, Orchardson & Collins 1987, Addy et al. 1987b, Minkoff & Axelrod 1987, Hansson et al. 1988).

In the first study patients were asked to rate their response to the various stimuli using a Visual Analogue Scale [continuous VAS], a 0-10 Numerical Rating VAS scale (NRS), and separate intensity verbal descriptor (IVD) and unpleasantness verbal descriptor (UVD) word scales (Duncan et al. 1989). During the study it was observed that patients initially preferred to give a numerical value rather than use the VAS, although there were no statistically significant differences between the two scoring systems. It may, therefore, be appropriate to have a training session with using the continuous VAS scale prior to commencement of a clinical study. One problem with the VAS is that it can only provide a unidimensional assessment of pain and as such cannot distinguish between the sensory, intensity and affective (unpleasantness) aspects of pain. Verbal descriptors on the other hand appear to provide a more sensitive tool for separating intensity and unpleasantness (Duncan et al. 1989). The present study would appear to support the conclusions of the Duncan et al. (1989) study, although the imprecise nature of the UVD words provided limited information in terms of accuracy and sensitivity, except at very low levels when assessing pain from CDS (**Figs.5.4.-5.8.**). The choice of the UVD words is therefore, important and consideration should be given to a more appropriate choice of word(s) for future studies. It should be recognised, however, that the handling of the data from patients in the two studies was different. As the data obtained from patients were derived from highly subjective assessments, an analysis of the overall trend of the results rather than detailed statistical analysis of isolated scores was advised (statistical advice). To this end a moving

average method was used in order to smooth out the otherwise inevitably erratic high and low points from the isolated scores (**Figs.5.4.-5.8.**). In the present study all four methods of assessment (using both mean scores and moving average analysis) demonstrated that patients perceived air stimulation (dental unit syringe) to cause the greatest discomfort and tactile sensation the least, which would appear to substantiate the sequence of application (e.g., tactile followed by air stimulation) in the 8- and 20- week studies.

The results of the first study would appear to confirm the conclusions of the Duncan et al.(1989) study that both verbal and non-verbal techniques quantify sensory and affective aspects of pain. The imprecise nature of UVD words, however, provided limited information in terms of accuracy or sensitivity, except at very low levels of discomfort, when assessing pain arising from CDS.

The McGill Pain Questionnaire (MPQ) has been used in various pain studies, although one main criticism is the complexity of the vocabulary. Several investigators (Hall et al.1986, Zakrzewska & Feinmann 1990) have reported that the MPQ is useful in diagnosis as well as monitoring treatment outcome, although Hansson et al.(1988) reported little correlation between the MPQ and other pain rating scales (VAS, VDS, & NRS).

In the second study, 40 patients from the 8-week study were shown 20 sets of words from a MPQ form (0 & 56 days) and asked to select a word (if applicable) from each set which best described their perception of pain or discomfort arising from CDS. The purpose of the study was not to monitor treatment outcome, but rather to observe whether the words chosen from the MPQ by patients on the first occasion would be similar to those chosen on the second occasion. As this study was designed to evaluate whether the words chosen by patients were reproducible between visits, the analysis was not based on obtaining a present pain index (PPI) or evaluating a pain rating index (PRI) etc.

Comparison of the recorded scores from patients demonstrated a very low percentage reproducibility (78/217 [36%]; 70/201 [34.8%])

respectively for both groups A & B). This does not suggest a particularly satisfactory relationship between the choice of words over the two visits and may consistently choosing a word to describe CDS. Each patient's contribution to the total number of scores was weighted. There were no significant differences between the two groups in the average number of recorded scores (6.5 [Gp A] & 5.7 [Gp B]). The contribution of each patient was, therefore, weighted as if they contributed that number of scores. For example, if a patient contributed 19 scores of which 9 were 'consistent', this is the equivalent of $9 \times 6.5/19 = 3.08$, if the total number of scores had been 6.5 instead of 19.

The number of times each word in each word set was most frequently mentioned, together with the overall popularity of each word quoted was also analysed (**Tables 5.7.-5.8., Fig.5.9.**). Words most commonly selected to describe pain from CDS, were sharp, tender, annoying, stabbing, aching and nagging.

The results of the second study demonstrated that patients were not consistent over the two visits concerning selection of word descriptors, although overall, the most frequently selected words, such as sharp, tender, shooting etc, described the characteristics of pain arising from CDS as reported by other investigators.

CHAPTER 6

Quantification of Thermal Stimuli

BIOMAT THERMAL PROBE STUDIES

Introduction

Problems in evaluating the clinical effectiveness of desensitizing agents appear to derive from a lack of satisfactory methods for evaluating the subjective response of the patient, which in turn is often modified by social, psychological and situational factors (McGrath 1986). Hence the variety of methods used to evaluate cervical dentinal sensitivity (CDS), for example, mechanical and thermal stimuli, together with patient subjective response to painful stimuli from daily experience (Minkov et al.1975, Green et al.1977, Tarbet et al.1979, 1980, 1982, Uchida et al.1980). Opinions vary as to the reliability of these various methods of assessment (Green et al.1977, Addy & Dowell 1983, Lecointre et al.1986, Addy et al.1987a).

Recently efforts have been made to develop controlled and reproducible stimuli more suited to the evaluation of CDS, for example, the Yeaple probe, the Yeh, Temptronic and thermo-electric devices (Silverman 1985, Minkoff & Axelrod 1987, Addy et al.1987a, Clark et al.1987, Person et al.1989).

This chapter reports the development of a thermal probe, a pilot model of which has been used for the assessment of CDS and nerve recovery studies (Ong 1983, Talhi et al.1985).

6.1. Materials and Methods

6.1.1. In vitro study (Probe calibration)

Calibration consisted of a series of 5 readings at set temperatures: 0, 4, 9, 14, 19, 24, 29, 34, 39, 44, 49, 54 and 59.9°C. The times taken to reach these temperatures were also recorded. The probe tip

temperatures were obtained from direct contact of a thermocouple attached to the tip and with direct recording from the BTP unit. Obtained and set temperatures were simultaneously recorded on a flat bed 4-pen recorder (Linseis GmbH, Cambridge, U.K.).

The Biomat Thermal Probe type TP3 (**Fig. 6.1.**), consists of a bench-mounted control unit containing power supplies and temperature control circuits, and a hand-held probe unit consisting of a brass pole assembly, which can be cooled or heated by a Peltier device heat pump. The rear face of the Peltier device is water-cooled, and an associated thermocouple measures probe temperature. The drive current for the Peltier device is supplied by a differential power amplifier which compares the output of the temperature sensing circuits with a reference voltage supplied by the set temperature circuits. The output current is dependent on the size of the error between the set and measured temperatures, and the gain of the amplifier is such that a fast rate of temperature change (0°C - 59.9°C) can be achieved with a minimum of overshoot. The thermocouple is connected to a thermocouple amplifier circuit with automatic cold junction compensation which drives the differential amplifier and a liquid crystal display (LCD) on the front panel of the control unit, as well as providing an analogue output for external use, e.g., chart recorder. The thermocouple amplifier also has an alarm circuit which detects thermocouple failure. The reference voltage is derived from a potentiometer circuit controlled by thumbwheel switches enabling the user to directly select the desired temperature (**Fig. 6.2.**). The unit operates from a 240 volt outlet and requires a water source to cool the Peltier device. The temperature range is 0°C - 59.9°C .

A series of in vitro consistency tests were performed with the BTP.

These included the following probe operations starting from a selected ambient temperature of 24°C .

- 1) From: 24°C - 59°C , 59°C - 24°C , 24°C - 0°C and 0°C - 24°C .
- 2) From: 24°C - 0°C and from 0°C - 24°C in 1°C and 5°C

Figure 6.1.(a)

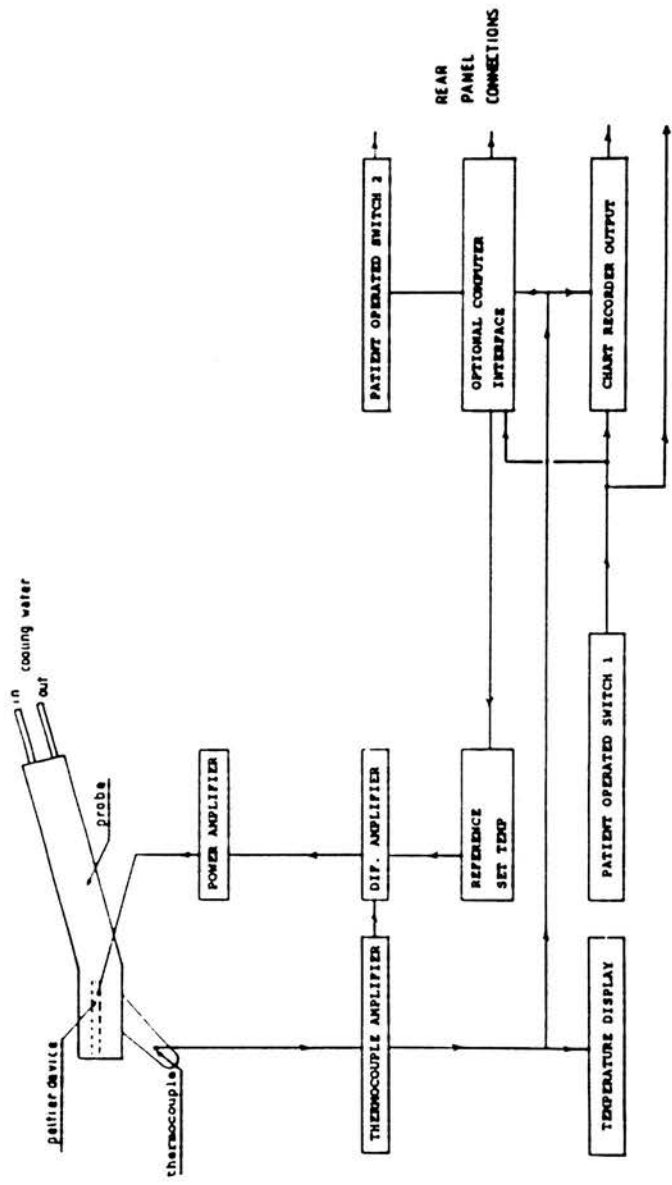


Biomat Thermal Probe unit
Figure 6.1.(b)



Biomat Thermal Probe tip

Figure 6.2.



Circuit diagram (Biomat Thermal Probe)

- 2) decrements and increments respectively.
- 3) From: 24°C-59°C and from 59°C-24°C in 1°C and 5°C increments and decrements respectively.

6.1.2. Clinical studies

Patients with cervical dentinal sensitivity (CDS) were enrolled in numbers sufficient to ensure a minimum of 10 patients for each of the studies (section 6.2.2.).

Inclusion and exclusion criteria were as follows:

Inclusion Criteria

Selection of patients was restricted to individuals with CDS, and investigator confirmation that they had cervical erosion, abrasion, and/or gingival recession on at least two non-adjacent teeth, sensitive to tactile and air stimulation and anterior to the second molar.

Teeth with restoration margins at least 5mm from the sensitive area were considered acceptable study teeth.

Exclusion Criteria

Patients with chronic systemic disease, such as diabetes, chronic debilitating disease, e.g., arthritis, history of allergy to dentifrices, of gingival surgery undertaken within the last six months or of teeth scaled during the previous month were excluded. Also excluded were patients taking analgesics, anti-convulsants, anti-histamines, sedatives, tranquillisers, mood altering or anti-inflammatory drugs. Patients who had active inflammatory periodontal disease judged by a gingival index score ≥ 2 at the mid-radicular site (Löe 1967), and individuals requiring extensive dental therapy or demonstrating gross oral neglect were also excluded, as were teeth with deep or defective restorations. Teeth used as abutments for partial dentures, or teeth with caries or cracked enamel or dentine were also excluded.

Screening

Following Joint Research and Ethics Committee approval and informed voluntary written subject consent, both medical and dental histories were reviewed and subjects asked to complete a questionnaire about their sensitivity complaint. Sensitive teeth were subsequently verified by the clinical investigator.

Selected subjects were examined first for tactile response (U.K. No. 6 probe) on the buccal or labial aspect of all teeth anterior to the second molar, subject to protocol restrictions (**See Inclusion & Exclusion criteria**). The location of the sensitive area on each responding tooth was also identified, together with any evidence of cervical erosion, abrasion and/or gingival recession.

Ten minutes later, the investigator assessed subject response to a one second application of cold air delivered from a dental unit syringe at 40-65 p.s.i. at a temperature of 19°C-24°C.

Procedure for Thermal Response (BTP)

Testing involved using a down/up/down method to determine the cold threshold of each sensitive tooth, commenced at 25°C and continued downwards in 5°C decrements until a positive response was obtained or, in the event of a continual negative response, until the end point of the range was obtained (0°C).

Once a positive response had been recorded the temperature was increased by 2°C and the tooth retested; if the response was negative the temperature was reduced by 1°C and testing continued until a second negative response was obtained.

The last positive response prior to two negatives was recorded as the first transition value.

Testing continued downwards until a positive response was recorded, the temperature was raised by 2°C and, if the response was negative, then the temperature was reduced by 1°C and the procedure continued as described above (second transition value). If, however, a positive

response was recorded at this temperature, then testing continued by 2°C increments until a negative response was obtained, and the procedure continued as above.

Testing continued until a third transition value was obtained, and the cold threshold value was calculated as the mean of all three transitions.

A VAS score form was also completed by the patient following stimulation with the BTP (see below).

An interstimulus interval of 30 seconds and 1 minute before each challenge were included in studies 1 & 3 and studies 2 & 4-5 respectively.

A second thermal test, namely cold air from a dental unit syringe was used for comparison in the studies (see below).

Procedure for assessing thermal response (cold air)

Ten minutes after BTP evaluation, a one second application of cold air (19°C-24°C) was applied from a dental unit syringe.

Patients indicated their response by placing a mark on a 10cm line (VAS), the distance from the 'no pain' end to this mark constituting a VAS score.

Study 1: Determination of interstimulus time interval

This study was designed to determine the time required to allow tooth temperature to revert to baseline prior to further stimulation. All sensitive teeth were evaluated. The baseline tooth temperature was recorded with a thermocouple (Comark Digital thermocouple 5000, Comark UK), and each tooth was then stimulated to the perceived threshold sensitivity for both hot and cold (BTP) following a 10 second application at set temperatures and a subsequent 1 second application of cold air from a dental unit syringe (19°C-24°C) after an interval of 10 minutes. The tooth temperature at the point at which sensitivity was felt was recorded, as well as the probe recording temperature at that point. Where no response was noted the end points for the hot and cold

range (0°C-59.9°C) were recorded (BTP). The difference between the two temperature readings and the time taken for the tooth temperature to return to baseline were calculated. For the cold air stimulus, however, the actual temperature at the point at which sensitivity was perceived by the patient was difficult to assess, since the air blast provides a variable temperature range (19-24°C) and, therefore, the tooth temperature before and on application, together with the time taken for the tooth temperature to revert to baseline, were recorded.

Study 2: Determination of the most effective method of stimulus presentation

This study was designed to determine the most appropriate method of presenting the thermal stimulus. One or more sensitive teeth per patient were tested. Both continuous (25°-0°C, 25°C-59.9°C) and 5°C decremental/incremental changes in temperature challenge were compared for reproducibility of threshold stimulation values. Threshold determination was by an up/down/up or down/up/down method (as described above) depending on whether hot or cold threshold was investigated. The interstimulus interval for presentation of the incremental stimulus was determined by study 1.

As problems were experienced initially with the time required to test patient response to a continuous application of cold (25-0°C), particularly at lower temperatures (< 5°C), it was decided to compare an obtained average (2 readings) from a single threshold stimulation value from decremental/incremental changes (5°C steps), together with hot and cold threshold stimulation values from continuous change (25°C-0°C and 25°C-59.9°C).

Study 3: Determination of change in sensitivity threshold following repeat application of the stimulus

This study was designed to determine whether repeated thermal stimulation with a minimum interstimulus time of 30 seconds produced either sensitization or desensitization of the tooth. One or more sensitive teeth per patient were subjected to repeated testing from the

BTP and the dental unit syringe.

The BTP was applied first to the test tooth (at the threshold stimulation temperature determined incrementally) for 10 seconds and repeated, following an interstimulus time interval of 30 seconds as determined by study 1, until no positive response was noted by the patient or until the threshold had been crossed 10 times. The number of applications up to and including this point was recorded. This was followed after a 10 minute interval by a one second application of cold air (dental unit syringe) and the procedure repeated as above. Patients were asked to respond (Yes/No) if they still perceived any discomfort from either of the two cold stimuli following repeated application. The sequence number at which this response occurred for both thermal stimuli was then compared.

A VAS score form was also completed for both methods of stimulus presentation at the onset and completion of testing.

Study 4: Determination of threshold stimulation temperature in non-sensitive teeth

This study was designed to determine the threshold stimulation temperature in non-sensitive teeth. Patients presenting with cervical erosion or abrasion and/or gingival recession, but who did not normally complain of CDS, or who experienced CDS in no more than 2 teeth, were recruited. Teeth which responded to the presenting stimuli were not included. All suitable teeth anterior to the second molar were tested.

Study 5: Reproducibility of threshold stimulation temperature values (BTP)

This study was designed to evaluate the reproducibility of the threshold stimulation values obtained from the BTP. Patients with at least one sensitive tooth were tested for hot and cold thresholds on two occasions (0 and 7 days).

The thermal challenge was decremental (5°C steps) commencing from 25°C, the interstimulus interval was one minute and the stimulus was applied for 10 seconds. A one second application of cold air from a dental unit

syringe was used for comparison. All temperature measurements (tooth and BTP) were recorded on a flat bed 4-pen recorder (Linseis GmbH Cambridge UK).

Patients were provided with a hand held control which enabled them to record stimulus onset and completion, as well as indicating a response when the stimulus was perceived within the 10 second application period.

Objective measurement of the thermal response was supplemented by patient subjective response utilising VAS forms.

6.1.3. Data analysis.

In vitro study

All data (obtained temperature values) were subjected to regression analysis. The results are shown in **Tables 6.1.-6.4., Figs. 6.3.-6.6..**

The time (actual values) required to obtain these set temperature values are shown in **Tables 6.5.-6.7..**

Clinical studies

All data were normally distributed and paired t-tests were, therefore, utilised to determine if apparent differences were statistically significant at the 95% confidence level.

6.2. Results

6.2.1. In vitro study (Probe calibration)

Correlation analysis between BTP tip and set temperatures gave a correlation coefficient of 0.99, which in turn indicated that 99.9% (r^2) of the variation in BTP tip temperature could be explained by variation in the set temperature. In other words, they were virtually identical. The maximum variation between BTP tip and set temperatures occurred at the extremes of the temperature range, and in neither case exceeded a difference of 2.2°C for cold and 2.7°C for hot respectively (**Tables 6.1.-6.4., Figs. 6.3.-6.6.**). 95% Confidence Intervals for

individual readings of the BTP digital display, tip temperatures and two recorder readings (y) were also calculated, and compared graphically with the set temperature line. **Figs. 6.3.-6.6.** show the optimum or set temperature gradient and the 95% Confidence Intervals for individual actual readings. At any given temperature there was less than a 5% chance that any individual recorded temperature would fall outside this range.

The time taken for the BTP to reach the set temperatures was consistent for all tests (5 readings). 1°C increments or decrements were accomplished within 1-3 seconds.

The time taken to reach the set temperatures from a selected ambient of 24°C to 59.9°C, 59.9°C-24°C, 24°C-0°C, 0°C-24°C and 0°C to 59.9°C, 59.9°C-0°C are shown in **Table 6.5..**

The time taken for 5°C increments/decrements from 24°C-59°C, 59°C-24°C and 24°C-4°C, 4°C-24°C are shown in **Tables 6.6.-6.7..**

There was little variation in time for the BTP to attain the set temperatures.

6.2.2. Clinical studies

All patients completed the study requirements and no 'drop outs' were experienced (**Table 6.8.**).

Study 1: Determination of interstimulus time interval

The initial temperature reading for the commencement of BTP evaluation was 37°C, but there was no response to the cold stimulus between 37°C and 25°C. It was, therefore, decided to commence testing (cold threshold stimulation) in all studies at 25°C. It was apparent, for hot threshold stimulation, that the BTP upper temperature range (25°C-59.9°C) was similarly unable to elicit any response in the majority of teeth tested. Therefore, determination of hot threshold stimulation was not generally evaluated.

The interstimulus time interval for BTP (36 teeth) and cold air (37 teeth) threshold evaluation was 30 seconds (**Tables 6.9.-6.10.**). For hot

Table 6.1.

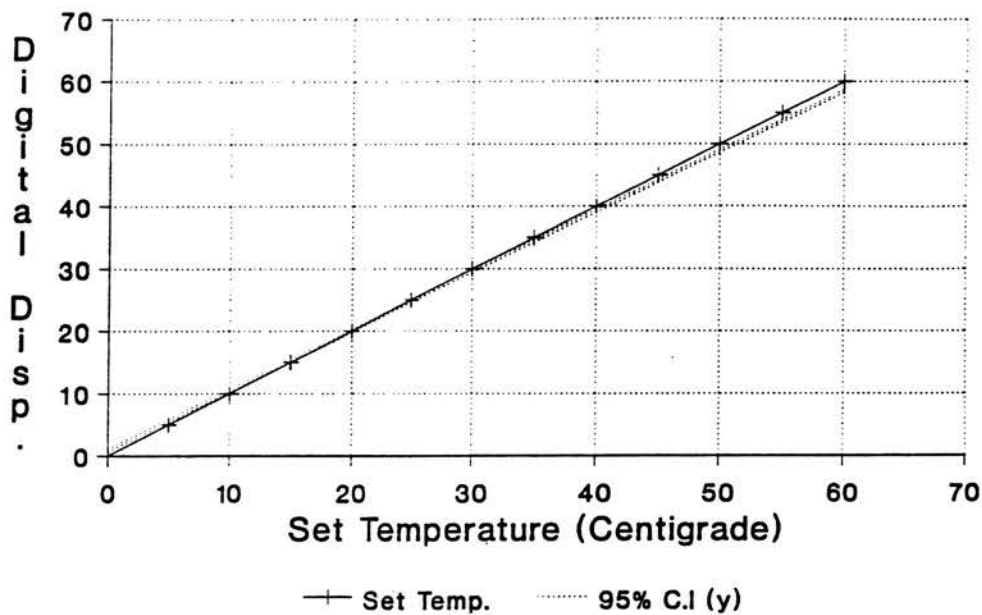
BIOMAT DIGITAL DISPLAY (°C)

x	y	SE (y)	95% CI (y)
0	0.8	0.1151	0.62 - 1.07
5	5.6	0.1147	5.42 - 5.87
10	10.4	0.1145	10.22 - 10.67
15	15.2	0.1143	15.03 - 15.48
20	20.1	0.1141	19.83 - 20.28
25	24.9	0.1140	24.63 - 25.08
30	29.7	0.1140	29.43 - 29.88
35	34.5	0.1141	34.23 - 34.68
40	39.3	0.1142	39.03 - 39.48
45	44.1	0.1143	43.84 - 44.28
50	48.9	0.1146	48.64 - 49.09
55	53.7	0.1149	53.44 - 53.89
60	58.5	0.1152	58.24 - 58.69

x = Set temperature (°C)

y = Observed temperature (°C)

Figure 6.3.



Biomat Digital Display readings (°C)
(Including 95% Confidence Intervals)

Table 6.2.

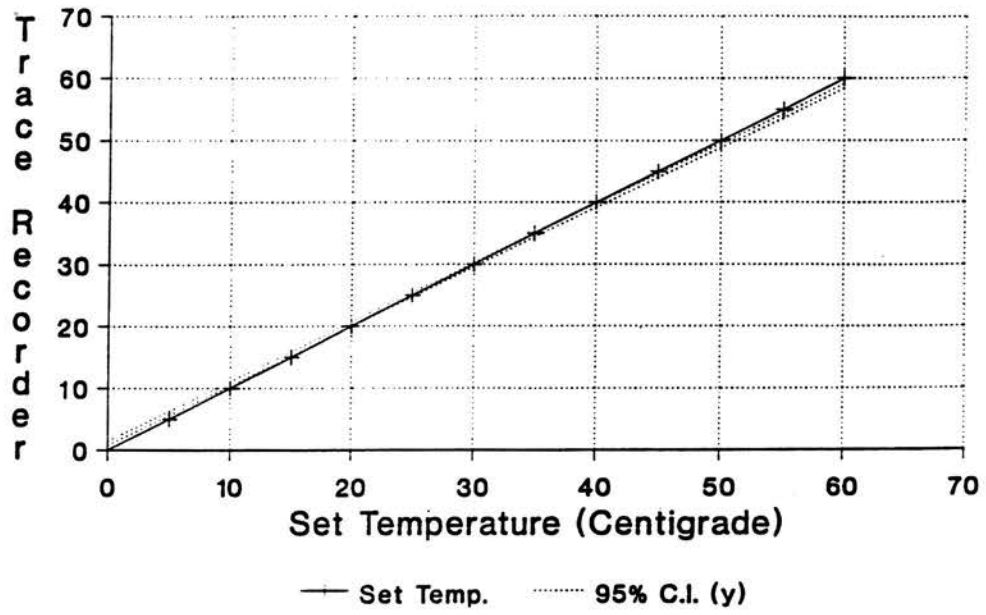
BIOMAT TRACE RECORDING (°C)

x	y	SE (y)	95% CI (y)
0	1.1	0.1785	0.75 - 1.45
5	5.9	0.1779	5.55 - 6.25
10	10.7	0.1775	10.36 - 11.06
15	15.5	0.1771	15.17 - 15.86
20	20.3	0.1769	19.96 - 20.67
25	25.1	0.1768	24.78 - 25.47
30	29.9	0.1767	29.58 - 30.28
35	34.7	0.1768	34.40 - 35.08
40	39.5	0.1769	39.20 - 39.89
45	44.3	0.1772	44.00 - 44.70
50	49.2	0.1776	48.81 - 49.50
55	54.0	0.1780	53.61 - 54.31
60	58.8	0.1786	58.42 - 59.12

x = Set temperature (°C)

y = Observed temperature (°C)

Figure 6.4.



Biomat Trace Recording readings (°C)
(Including 95% Confidence Intervals)

Table 6.3.

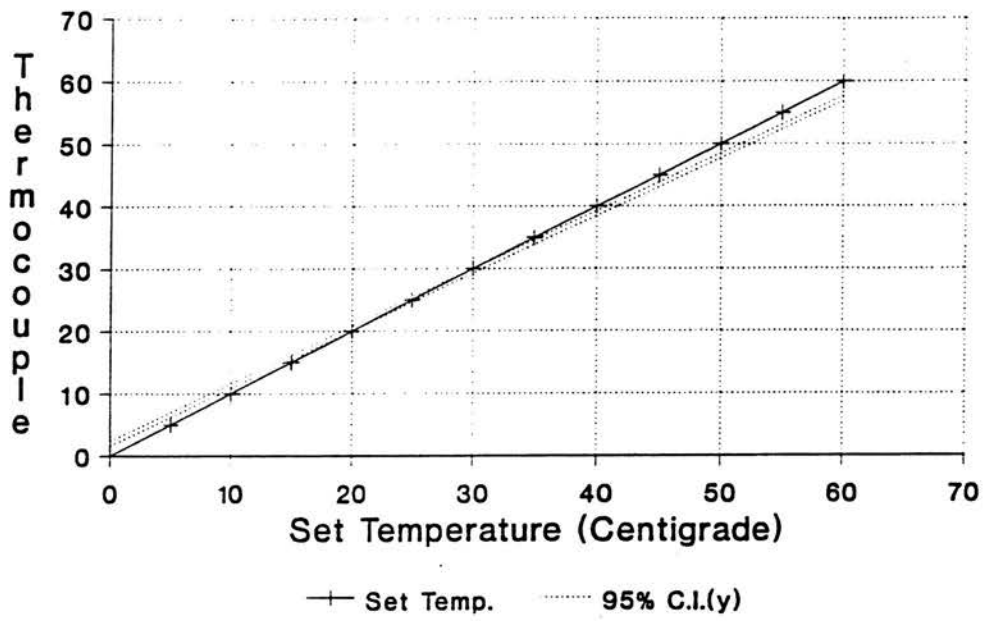
BIOMAT PROBE TIP (°C)
(Thermocouple)

x	y	SE (y)	95% CI (y)
0	2.0	0.1984	1.59 - 2.36
5	6.6	0.1979	6.20 - 6.96
10	11.2	0.1974	10.81 - 11.59
15	15.8	0.1970	15.43 - 16.20
20	20.4	0.1968	20.04 - 20.81
25	25.0	0.1966	24.65 - 25.42
30	29.6	0.1966	29.26 - 30.03
35	34.3	0.1966	33.88 - 34.65
40	38.9	0.1968	38.49 - 39.26
45	43.5	0.1971	43.10 - 43.87
50	48.1	0.1976	47.11 - 48.49
55	52.7	0.1980	52.32 - 53.10
60	57.3	0.1986	56.93 - 57.71

x = Set temp (°C)

y = Observed temp (°C)

Figure 6.5.



Biomat Probe Tip readings (°C)
(Thermocouple)

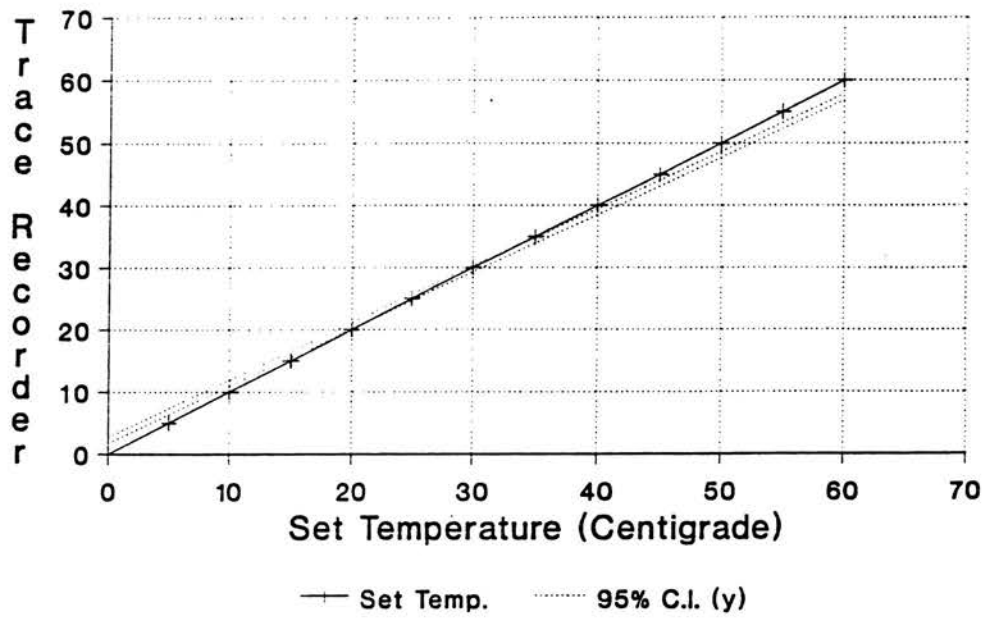
Table 6.4.BIOMAT TIP TRACE RECORDING (°C)

x	y	SE (y)	95% CI (y)
0	2.2	0.2503	1.74 - 2.72
5	6.8	0.2495	6.34 - 7.32
10	11.4	0.2489	10.93 - 11.91
15	16.0	0.2484	15.53 - 16.50
20	20.6	0.2481	20.12 - 21.10
25	25.2	0.2479	24.72 - 25.69
30	29.8	0.2487	29.31 - 30.28
35	34.4	0.2479	33.91 - 34.88
40	39.0	0.2482	38.50 - 39.47
45	43.6	0.2485	43.09 - 44.07
50	48.2	0.2490	47.69 - 48.66
55	52.8	0.2497	52.28 - 53.26
60	57.4	0.2505	56.87 - 57.86

x = Set temp (°C)

y = Observed temp (°C)

Figure 6.6.



Biomat Probe tip trace recorder readings (°C)

Table 6.5.(a)

Time taken to obtain set temperatures (24°C - 59.9°C, 59.9°C - 24°C; 24°C - 0°C, 0°C - 24°C; 0°C - 59.9°C, 59.9°C - 0°C (seconds)

Temp setting °C	BTP unit (seconds)	BTP tip (seconds)
24 - 59.9°C	42:42:42:42:42:	60:60:60:60:60:
59.9 - 24°C	42:42:42:42:42:	60:60:60:60:60:
24 - 0°C	120:126:126:126:132	120:138:132:138:138
0 - 24°C	18:18:18:18:18:	60:60:60:60:60:
0 - 59.9°C	60:60:60:60:60:	78:78:78:78:78:
59.9 - 0°C	240:186:210:204:216	222:192:204:198:204

Table 6.5.(b)

Summary: Time taken to obtain a set temperature range (seconds)

Temp setting °C	BTP unit (seconds)	BTP tip (seconds)
24 - 59.9°C	42	60
59.9 - 24°C	42	60
24 - 0°C	126	133.2
0 - 24°C	18	60
0 - 59.9°C	60	78
59.9 - 0°C	211.2	204

Table 6.6.(a)

Time taken to obtain set temperatures (24°C - 59.9°C and 59.9°C - 24°C in 5°C increments/decrements (seconds)

Temp setting °C	BTP unit (seconds)	BTP tip (seconds)
24 - 29°C	6:6:6:6:6:	18:18:18:18:18:
29 - 34°C	6:6:6:6:6:	18:18:18:18:18:
34 - 39°C	6:6:6:6:6:	18:18:18:18:18:
39 - 44°C	6:6:6:6:6:	18:18:18:18:18:
44 - 49°C	6:6:6:6:6:	18:18:18:18:18:
49 - 54°C	6:6:6:6:6:	18:18:18:18:18:
54 - 59°C	6:6:6:6:6:	18:18:18:18:18:
59 - 54°C	6:6:6:6:6:	18:18:18:18:18:
54 - 49°C	6:6:6:6:6:	18:18:18:18:18:
49 - 44°C	6:6:6:6:6:	18:18:18:18:18:
44 - 39°C	6:6:6:6:6:	18:18:18:18:18:
39 - 34°C	6:6:6:6:6:	18:18:18:18:18:
34 - 29°C	6:6:6:6:6:	18:18:18:18:18:
29 - 24°C	6:6:6:6:6:	18:18:18:18:18:

Table 6.6.(b)

SUMMARY: Time taken to obtain set temperatures (24°C - 59.9°C and 59.9°C - 24°C) in 5°C increments/decrements (seconds)

Temp Setting °C	BTP unit (seconds)	BTP tip (seconds)
24 - 29°C	6	18
29 - 34°C	6	18
34 - 39°C	6	18
39 - 44°C	6	18
44 - 49°C	6	18
49 - 54°C	6	18
54 - 59.9°C	6	18
59.9 - 54°C	6	18
54 - 49°C	6	18
49 - 44°C	6	18
44 - 39°C	6	18
39 - 34°C	6	18
34 - 29°C	6	18
29 - 24°C	6	18

Table 6.7.(a)

Time taken to obtain set temperature (24°C - 0°C and 0°C - 24°C) in 5°C increments/decrements (seconds)

Temp setting °C	BTP unit (seconds)	BTP tip (seconds)
24 - 19°C	6: 6: 6: 6: 6:	18:18:18:18:18:
19 - 14°C	12:12:12:12:12:	24:18:24:24:18:
14 - 9°C	18:18:18:18:18:	30:18:24:24:30:
9 - 4°C	30:30:30:30:30:	36:24:36:30:33:
4 - 9°C	6: 6: 6: 6: 6:	18:18:18:18:18:
9 - 14°C	6: 6: 6: 6: 6:	18:18:18:18:18:
14 - 19°C	6: 6: 6: 6: 6:	18:18:18:18:18:
19 - 24°C	6: 6: 6: 6: 6:	18:18:18:18:18:

Table 6.7.(b)

Summary: Times taken to obtain set temperatures (24°C - 4°C and 4°C - 24°C) in 5°C increments/decrements (seconds)

Temp setting °C	BTP unit (seconds)	BTP tip (seconds)
24 - 19°C	6	18
19 - 14°C	12	22.8
14 - 9°C	18	26.4
9 - 4°C	30	34.2
4 - 9°C	6	18
9 - 14°C	6	18
14 - 19°C	6	18
19 - 24°C	6	18

Table 6.8.Patient Data

Study No	N	F	M	Mean Age (SD)
1	14	8	6	44.3 (5.94)
2.1	11	5	6	43.8 (6.58)
2.2	10	7	3	43.3 (6.32)
3	11	5	6	43.8 (6.58)
4	10	7	3	46.2 (12.32)
5	11	5	6	43.8 (6.58)

Key

N = Number

F = Female

M = Male

threshold (40 teeth), however, the BTP upper limit of 59.9°C did not, for the majority of teeth tested, identify a threshold value. Therefore the upper limit was used to determine the interstimulus interval which proved to be within 18 seconds for most readings (Table 6.11.).

The tooth surface temperature was measured throughout the study by means of a thermocouple situated approximately 1mm from the BTP.

Study 2: Determination of the most effective method of stimulus presentation

Cold threshold stimulation temperature

Paired t-tests were used to determine whether or not there were statistically significant differences between mean probe scores (°C) of the two methods of presentation in each of the two studies (5°C decremental vs. average threshold values [2 readings] and 5°C decremental vs. continuous [25°C-0°C]) (Tables 6.12.-6.13.).

No significant differences were demonstrated for the 5°C decremental vs. average threshold presentation values [39 teeth] or for 5°C decremental vs. continuous presentation [30 teeth], although the latter, however, approached significance (Table 6.14.).

No statistically significant difference was demonstrated for VAS scores in the 5°C decremental vs. continuous presentation study (Table 6.14.).

Hot threshold stimulation temperature

Thirty four teeth were stimulated using the BTP. The investigator was unable to determine an average threshold stimulation value for hot stimulation within the temperature range of the BTP when comparing 5°C increments and average threshold values. Comparison between 5°C incremental vs. continuous methods of stimulus presentation also proved difficult and consequently the upper limits of the temperature range were reached in the majority of teeth with no perception of discomfort, irrespective of the method of presentation. No differences were demonstrated in VAS scores.

Table 6.9.

Determination of interstimulus time interval Biomat Thermal Probe (Cold)

Patient No	Baseline Tooth Temp (°C)	Application Tooth Temp (°C)	Stimulation Temp (°C)	Time (sec)
1	33.0	22.0	10.3	30
	34.0	27.9	20.2	24
2	32.0	26.5	3.1	30
	33.0	27.0	17.1	30
3	32.0	12.0	13.0	30
	32.0	20.0	23.0	30
4	32.0	25.0	16.3	30
	34.0	23.0	14.4	30
5	33.0	26.0	17.2	30
	32.5	28.0	3.0	24
	33.0	26.5	10.5	30
6	34.0	30.5	13.4	15
	33.0	27.5	21.0	15
	34.0	28.0	1.8	21
7	32.0	30.0	10.5	17
	34.0	30.0	20.1	30
8	33.0	29.0	3.4	30
	32.0	28.0	4.1	30

Key to Tables 6.9.-6.11.

Baseline Tooth temperature:

The recorded surface tooth temperature at the commencement of testing.

Application Temperature:

The recorded surface tooth temperature when the BTP was applied at the established threshold value.

Stimulation Temperature:

The recorded temperature (BTP) at which the patient perceived sensation when the tooth was stimulated (Threshold value).

Time:

The time (seconds) taken for the surface tooth temperature to return to baseline values after application of the stimulus.

Table 6.9.(cont.)

Patient No	Baseline Tooth Temp (°C)	Application Tooth Temp (°C)	Stimulation Temp (°C)	Time (sec)
9	32.0	20.0	10.5	15
	32.0	20.0	10.5	30
10	32.0	27.0	2.5	21
	33.0	25.0	14.3	21
	33.0	28.0	5.9	24
11	32.0	18.0	3.1	30
	32.0	20.0	10.5	30
12	34.0	17.0	5.9	30
	34.0	26.0	2.6	27
	34.0	26.0	2.0	30
	33.0	26.0	2.6	30
13	32.0	28.0	5.7	12
	34.0	18.0	2.5	21
14	32.0	24.0	10.3	30
	30.0	12.0	2.7	30
	30.0	23.0	1.9	27
	31.0	24.0	2.0	30
	31.0	12.0	2.9	30

Table 6.10.Determination of interstimulus time interval (cold air)

Patient No	Baseline Tooth Temp (°C)	Application Tooth Temp (°C)	Time (Sec)
1	32.0	22.0	30
	32.0	20.2	30
2	32.0	19.0	21
	32.0	19.9	21
3	32.0	20.0	21
	32.0	20.0	24
4	34.0	29.0	21
	33.7	27.0	30
5	34.0	23.9	30
	32.0	22.0	30
	33.0	23.0	30
6	----	-----	--
7	33.5	25.0	30
	34.0	23.9	30
8	35.0	25.0	15
	33.0	25.5	30
9	32.0	24.0	15
	32.0	23.9	30

Table 6.10.(cont.)

Patient No	Baseline Tooth Temp (°C)	Application Tooth Temp (°C)	Time (Sec)
10	32.0	24.0	30
	33.0	26.0	30
	31.5	25.9	24
11	33.0	21.0	27
	33.0	23.5	30
12	34.0	21.0	30
	34.0	24.0	30
	34.0	20.0	15
	34.0	24.0	30
13	33.0	27.0	30
	34.0	25.0	27
	32.0	25.0	18
	32.0	26.0	30
14	32.0	22.0	24
	33.0	24.0	30
	30.0	19.0	30
	31.0	18.5	30
	30.0	21.5	27
	31.0	22.0	24
	31.0	26.5	15

Table 6.11.

Determination of interstimulus time interval Biomat Thermal Probe (heat)

Patient No	Baseline Tooth Temp (°C)	Application Tooth Temp (°C)	Stimulation Temp (°C)	Time (Sec)
1	33.9	42.2	58.6	30
	35.0	38.0	58.4	15
2	33.0	38.0	57.0	15
	33.0	36.0	58.2	15
3	34.0	41.0	58.4	30
	34.0	40.0	58.4	30
4	34.0	36.0	57.6	15
	34.0	36.0	42.0	15
5	34.0	36.0	56.7	15
	34.0	36.0	58.4	15
	35.0	37.0	58.4	15
6	34.0	35.9	58.2	12
	34.0	35.9	44.8	12
	34.0	35.0	58.1	12
7	34.0	38.5	58.1	12
	34.0	43.8	57.1	30
8	33.0	35.0	58.4	12
	33.5	41.0	58.5	15

Table 6.11.(cont.)

Patient No	Baseline Tooth Temp (°C)	Application Tooth Temp (°C)	Stimulation Temp (°C)	Time (sec)
9	32.0	35.0	58.2	12
	32.0	34.0	58.2	12
10	34.0	36.0	58.5	15
	34.0	36.0	58.5	15
	34.0	35.0	58.5	12
11	31.0	34.5	58.5	15
	32.0	35.0	58.4	15
12	33.0	35.0	58.3	12
	35.0	38.0	58.3	15
	35.0	39.0	58.3	15
	34.0	36.0	58.3	12
13	34.0	37.0	58.5	12
	34.0	36.5	58.5	12
	34.0	36.0	58.6	15
	32.0	35.0	58.5	15
14	34.0	42.0	58.5	21
	29.0	32.0	58.5	12
	28.0	30.0	58.5	12
	30.0	34.0	58.5	12
	31.0	32.0	58.5	12
	33.0	40.0	58.5	12
	34.0	42.0	58.5	21

Table 6.12.

Incremental vs. Average threshold values (°C) Biomat Thermal Probe (cold)

Patient No	Incremental Threshold Temp (°C)	Average Threshold Temp (°C)	Threshold Temp (°C)	
			1	2
1	8.6	7.7	8.6	6.8
	7.2	6.6	6.8	6.3
2	14.4	16.8	16.3	17.3
	17.3	18.0	17.3	18.7
	20.1	21.3	21.1	21.5
3	10.6	10.6	10.6	10.6
	5.0	5.0	5.0	5.0
4	11.1	11.0	10.8	11.1
	10.0	10.0	10.0	10.0
5	17.2	17.4	17.5	17.2
	3.0	3.3	3.5	3.0
	10.5	11.6	12.0	11.1
	15.4	15.9	15.4	16.3
	5.1	3.6	4.0	3.2
7	9.7	8.8	8.8	8.7
	17.3	14.9	14.4	15.3
8	5.9	6.9	7.8	5.9
	5.9	6.8	7.7	5.9
	4.0	2.4	2.4	2.3
	14.3	14.1	13.9	14.3
	4.0	3.6	4.0	3.1
9	10.5	11.7	11.5	11.9
	10.5	11.2	11.9	10.5
	2.1	2.2	2.1	2.3
	11.5	10.1	10.5	9.6
10	2.7	2.5	4.0	4.9
	5.9	6.4	6.9	5.9
11	3.1	2.7	3.2	2.2
	10.5	11.1	11.5	10.6

Table 6.12.(cont.)

Patient No	Incremental Threshold Temp (°C)	Average Threshold Temp (°C)	Threshold Temp (°C)	
			1	2
	5.9	6.4	6.3	6.4
12	5.2	7.3	5.8	8.7
	2.0	2.7	3.1	2.3
	3.7	5.5	6.0	5.0
	10.6	9.7	10.6	9.2
	2.4	2.6	3.1	2.0
11	2.9	2.2	2.3	2.1
	2.9	2.5	2.6	2.4
4	5.8	5.4	5.9	4.9
	15.0	16.4	16.9	15.9

Table 6.13.

Continuous vs. incremental method of presentation Biomat Thermal Probe scores (cold)

Patient No	Continuous Threshold Temp (°C)	Incremental Threshold Temp (°C)	Cont ¹ /Incremental ² VAS Scores (cm)	
			1	2
1	5.8	8.6	1.1	0.5
	7.7	7.2	1.7	1.4
2	14.8	14.4	0.5	1.1
	9.1	20.7	1.9	0.4
3	2.4	10.6	0.0	1.0
	2.4	5.0	0.0	1.1
4	10.1	5.8	3.7	0.9
	13.6	15.0	3.7	3.7
5	2.2	11.5	0.0	5.0
	2.1	2.2	0.0	4.5
	12.0	11.2	4.3	4.3
8	3.4	2.7	1.2	1.2
	4.1	5.9	1.2	1.2
11	2.9	3.1	0.6	0.6
	4.9	10.5	2.5	2.5
	4.2	5.9	0.0	0.2
	4.3	2.4	0.0	0.3
12	2.0	5.2	4.0	1.0
	2.7	2.0	0.0	0.3
	3.7	2.9	0.0	0.6
	5.3	5.9	0.3	0.3
13	12.1	10.1	2.6	2.6
	11.0	10.2	3.3	3.3
11	3.4	2.9	2.9	4.6
	3.0	2.9	0.0	4.6
14	10.3	13.9	4.6	2.9
	2.7	2.6	0.0	2.5
	1.9	2.0	0.0	0.0
	2.0	1.8	0.0	2.7
	2.9	1.9	0.0	3.8

Table 6.14.

Summary: Determination of the most effective method of stimulus presentation

Study No	Difference between means	t value (df)	p value	95% C.I. of the mean
2.1	-0.13	0.80 (38)	0.4304	-0.46-0.20
2.2	-1.3	2.0 (29)	0.0548	-2.56-0.03
2.2*	-0.63	1.77 (29)	0.0865	-1.36-0.10

Key

2.1 5°C decremental vs. average threshold values (°C)

2.2 5°C decremental vs. continuous presentation (°C)

2.2* VAS scores (cm) for 5°C decremental vs. continuous presentation

Study 3: Determination of change in sensitivity threshold following repeat application of the stimulus

Twenty six teeth were subjected to thermal stimulation from BTP and cold air from a dental unit syringe. Repeated application of cold following a 30 second time interval resulted in a transient desensitization of the tooth in the majority of teeth tested. Only one tooth retained a positive response throughout the study (Tables 6.15.-6.16.).

This was verified by VAS score which indicated a small but consistent level of perceived discomfort following repeated application of cold (BTP).

Paired t-tests were used to determine whether or not there were statistically significant differences between the mean scores from the two methods of thermal presentation (Table 6.17.).

No difference between VAS for BTP and cold air was observed at the completion of the test.

Study 4: Determination of threshold stimulation temperature in non-sensitive teeth

The cold threshold stimulation temperature was determined for 137 non-sensitive teeth following stimulation with BTP (Table 6.18.). The mean was 0.45°C (\bar{x} = 0.30 excluding outlier) (95% C.I. 0.04 to 0.56, Median = 0.0).

By way of comparison a group of 11 patients (26 sensitive teeth) from study 5 demonstrated a mean of 8.4°C (95% C.I. for the mean, 5.83 to 10.91, Median = 5.9) (Table 6.19.).

Pearson's Correlation Coefficient for non-sensitive teeth BTP threshold stimulation temperature and VAS was 0.5231 (r^2 = 0.2736, i.e. 27.4% of the variation in VAS can be explained by variation in BTP threshold stimulation temperature values).

This does not suggest a particularly close relationship between these two variables.

Study 5: Reproducibility of threshold stimulation temperature

Comparison of cold, hot (BTP) and cold air threshold stimulation temperature values and VAS scores from 26 teeth (**Tables 6.19.-6.21.**) demonstrated no statistically significant differences between the two examination visits (**Table 6.22.**).

Percentage reproducibility of BTP threshold temperature stimulation indicated that 19/26 teeth (73.1%), had differences $< 5^{\circ}\text{C}$, and 7/26 teeth (26.9%), had differences $> 8^{\circ}\text{C}$.

Table 6.15.

Determination of change in sensitivity threshold following repeated stimulation (cold air)

Patient No	Threshold stimulation value*	Threshold Stimulation value ¹ VAS (cm)	Threshold Stimulation value, VAS (cm)
1	4	0.7	0.0
	8	1.4	0.0
2	1	3.2	0.0
	7	10.0	0.0
3	2	0.3	0.0
	3	0.6	0.0
4	5	6.3	0.0
	5	1.1	0.0
5	4	7.6	0.0
	3	4.1	0.0
	6	4.7	0.0
7	2	3.7	0.0
	1	7.0	0.0
8	3	2.6	0.0
	8	7.7	0.0
9	3	3.5	0.0
	2	2.1	0.0
10	6	2.2	0.0
	3	1.0	0.0
	3	4.2	0.0
11	2	1.1	0.0
	7	2.6	0.0
12	4	2.5	0.0
	5	2.4	0.0
	8	5.1	0.0
	6	4.0	0.0

* Last positive value following stimulation

¹ VAS score (cm) at last positive value

² VAS score (cm) at end of stimulation

Table 6.16.

Determination of change in sensitivity threshold following repeated stimulation (Biomat Thermal Probe scores: cold)

Patient No	Stimulation Threshold Value*	Stimulation Threshold Value ¹ VAS (cm)	Stimulation Threshold Value ² VAS (cm)
1	2	0.9	0.0
	2	0.4	0.0
2	10	1.1	0.0
	4	0.4	0.0
3	2	1.0	0.0
	2	1.1	0.0
4	7	0.9	0.0
	6	3.7	0.0
5	3	5.0	0.0
	3	4.5	0.0
	3	4.3	0.0
7	2	0.2	0.0
	2	1.0	0.0
8	4	1.2	0.0
	5	1.2	0.0
9	2	0.8	0.0
	5	0.3	0.0
10	2	0.5	0.0
	2	2.1	0.0
	2	1.6	0.0
11	4	0.6	0.0
	4	2.5	0.0
12	-	---	---
	-	---	---
	4	1.3	0.0
	3	1.0	0.0

* Last positive value following stimulation

¹ VAS score (cm) at last positive value

² VAS score (cm) at end of stimulation

Table 6.17.

SUMMARY: Determination of change in sensitivity threshold following repeat application of the stimulus

BTP/cold air scores	Difference between means	t value (df)	p value	95% C.I. for the mean
BTP/cold air	-0.70	1.15 (23)	0.2637	-1.99-0.57
BTP	1.52	5.20 (23)	0.0000	0.91-2.13
cold air	3.53	7.15 (25)	0.0000	2.51-4.54
BTP/cold air	2.05	3.16 (23)	0.0015	0.87-3.22

Key

BTP/cold air = Difference in sequence response following stimulus application

BTP = VAS scores (cm) on onset and completion of stimulus application

cold air = VAS scores (cm) on onset and completion of stimulus application

BTP/cold air = VAS scores (cm) on commencement of stimulus application

Table 6.18.

Determination of threshold in non-sensitive teeth Biomat Thermal Probe
Threshold scores (cold)

[illegible]

Table 6.18.(cont.)

Patient No	Temp (°C)	VAS (cm)	Patient No	Temp (°C)	VAS (cm)
6	0.0	0.0	8	0.0	0.0
	0.0	0.5		0.0	0.0
	0.0	4.0		0.0	0.0
	0.0	0.0		0.0	0.0
	5.0	3.3		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	3.3		0.0	0.0
	0.0	3.3		0.0	0.0
	0.0	0.5		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	0.0		0.0	0.0
7	0.0	0.0		0.0	0.0
	0.0	0.0	9	0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	4.6		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	0.0		15.0	4.9
	0.0	7.6		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	0.0		0.0	0.0
8	0.0	0.0		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	0.0		0.0	0.0
	0.0	0.0		0.0	0.0

Table 6.18. (cont.)

[illegible]

Table 6.19.Reproducibility study Biomat Thermal Probe Threshold scores (cold)

Visit 1		Visit 2	
(°C)	VAS (cm)	(°C)	VAS (cm)
10.3	2.2	2.0	2.9
20.2	0.4	10.5	0.3
3.1	2.1	1.6	0.0
17.1	2.2	17.3	1.0
1.0	3.0	10.6	1.0
4.0	1.5	5.0	1.1
16.3	1.8	11.1	1.8
14.4	2.3	10.0	7.4
17.2	7.1	18.9	5.0
3.0	1.1	2.2	4.5
10.5	3.2	21.8	5.5
10.5	2.3	9.7	0.2
20.1	0.1	17.3	1.0
2.0	1.0	2.7	1.2
5.9	1.3	5.9	1.2
10.5	0.3	10.5	0.4
2.1	0.3	4.0	0.3
5.9	4.1	5.9	0.5
2.5	2.7	4.0	1.6
14.3	0.2	4.0	2.1
3.1	0.6	5.9	0.2
10.5	2.5	2.4	0.3
5.9	0.5	5.2	0.7
2.6	0.0	2.0	0.3
2.0	0.0	3.7	0.6
2.6	0.0	10.6	0.3

Table 6.20.Reproducibility study Biomat Thermal Probe Threshold Scores (heat)

Visit 1		Visit 2	
°C	VAS (cm)	°C	VAS (cm)
58.6	0.7	58.5	0.0
58.4	0.0	58.5	0.0
57.0	5.5	58.4	0.0
58.2	1.5	52.9	6.0
58.4	0.0	58.5	0.0
58.4	0.0	58.5	0.0
57.6	0.9	58.2	2.8
42.0	6.5	56.3	6.3
56.7	0.0	58.2	0.0
58.4	0.0	58.2	0.0
58.4	0.0	58.2	0.0
58.1	0.0	58.4	0.0
57.1	0.5	58.4	0.0
58.5	0.0	58.5	0.0
58.4	0.0	58.4	0.0
58.2	0.5	58.3	0.5
58.2	0.0	58.3	0.0
58.5	0.0	58.5	0.0
58.5	0.0	58.0	0.0
58.5	0.0	58.0	0.0
58.5	0.0	58.6	0.0
58.4	0.0	58.6	0.0
58.3	0.0	58.3	1.3
58.3	0.0	58.3	0.0
58.3	0.0	58.3	0.0
58.3	0.0	58.3	0.0

Table 6.21.Reproducibility study (Biomat Thermal Probe) Cold Air VAS Scores (cm)

Visit 1	Visit 2
2.1	0.8
1.9	4.1
4.2	2.2
6.5	8.5
1.5	0.3
1.1	0.6
3.2	2.9
2.9	3.2
6.9	4.5
2.1	3.2
2.1	3.3
4.5	0.7
3.5	7.0
2.4	2.6
6.0	7.6
3.1	1.3
2.4	0.2
3.7	2.2
4.4	6.3
4.4	7.3
1.1	1.4
2.6	1.4
3.4	3.3
1.3	2.5
2.9	4.3
4.6	4.6

Table 6.22.SUMMARY: Reproducibility of threshold stimulation temperature

Mode of Stimulus	Difference between means	t value (df)	p value	95% C.I. for the mean
BTP (cold)	0.49	0.48 (25)	0.6373	-1.63-2.62
BTP (cold)*	0.054	0.15 (25)	0.8837	-0.70-0.80
BTP (hot)	0.51	0.86 (25)	0.3972	0.72-1.75
cold air	-0.058	0.16 (25)	0.8717	-0.79-0.67

Key

BTP (cold) = Temperature values (°C)

BTP (cold)* = VAS score values (cm)

BTP (hot) = Temperature values (°C)

cold air = VAS score values (cm)

Discussion

The original intension of the in vitro and in vivo probe studies was to compare the BTP with an air delivery system (Pearson et al. 1989). Unfortunately due to performance problems with this device, the studies were unable to proceed in their original format. A dental unit syringe (cold air blast) was subsequently included for comparison with the BTP in the clinical studies.

The results of the in vitro probe calibration studies would indicate that the BTP was both accurate and consistent over the temperature range 0°C- 59.9°C. The digital display temperature (**Fig. 6.3.**) appeared to be the most accurate and consistent when compared to the other temperature readings (**Figs.6.4.-6.6.**), although these differences too were negligible. This would indicate, for all practical purposes, that for any given temperature setting the digital display and BTP tip temperatures were virtually identical. In other words, the set temperature reading as indicated on the digital display would accurately reflect the actual temperature registered at the BTP tip. Overall, the results of the in vitro probe calibration study show remarkable consistency, both in temperature measurement and in times required for the BTP to reach set temperatures (**Tables 6.1.-6.7., Figs.6.3.-6.6.**).

The results of the first clinical study would indicate that recorded tooth temperatures reverted back to baseline values within 15-20 seconds following heat stimulation and 30 seconds for cold (**Table 6.9.-6.11.**). From this study it would appear that these recorded temperature changes were localised to a very small area of the exposed root surface. It is unlikely that the actual tooth temperature would have risen/decreased dramatically during the short application period of 10 seconds (Cp Grüsser et al 1982). Following the results of this study a one minute time interval for most of the remaining studies with the exception of study 3.

The results for the second study showed no significant differences

between the methods of presentation of the stimulus (**Table 6.14.**), although from a practical viewpoint a 5°C incremental/decremental change appeared to be more satisfactory for both the patient and the investigator. There may also be greater problems with a continuous presentation approach e.g., conditioning of the tooth as well as maintaining BTP tip temperatures in the mouth.

The problem of repeated stimulation was addressed in the third study and it would appear that repeated thermal stimulation (BTP/cold air blast) with a 30 second time interval resulted in desensitization of the tooth. There were no significant differences between the two methods of thermal presentation (**Table 6.17.**).

The results of the fourth study demonstrated that non-sensitive teeth do not respond to tactile or thermal stimuli, whereas sensitive teeth generally respond to either or both stimuli (**Tables 6.18.-6.19.**). This observation may be of interest in the discussion as to whether teeth which respond to various stimuli are in actual fact 'hypersensitive'. It would be possible that such teeth have underlying and undetected pulpal pathology which may, according to Kim and co-workers (1990, 1992), lower the sensory nerve excitability threshold to tactile and/or thermal stimulation, thereby eliciting a response from the patient. Trowbridge & Silver (1990), however, have stated that evidence to support such a hypothesis is lacking.

Comparison of BTP and cold air scores over the two visits (study 5) demonstrated that there were no statistically significant differences (**Table 6.22.**) and would indicate that the BTP was reproducible in recording accurate temperature thresholds and compared favourably with the report of Pearson *et al.* (1989) using an air delivery system.

It should be mentioned that hot thresholds were outside the upper limit of the BTP temperature range and, while the BTP can be adjusted to enable precise measurement of this threshold, testing teeth with temperatures which lie outside this range (> 60°C) may induce detrimental changes in the tooth.

The results of the clinical studies would, therefore, indicate that

the BTP was well tolerated by patients and appeared to be both accurate and consistent in the measurement of cold threshold stimulation temperatures. Overall the BTP appeared to provide a more objective method of assessing thermal (cold) sensitivity than cold air from a dental unit syringe. It was concluded, on the basis of these studies that the BTP would be useful as a means of objectively assessing patient response to thermal stimuli in clinical studies designed to evaluate the efficacy of desensitizing agents.

CHAPTER 7Discussion

There are problems in evaluating the efficacy of desensitizing dentifrices in clinical trials. Opinions also vary as to the variation and validity of the methods of assessment used to evaluate these dentifrices (**section 1.3.**). G.V. Black (1908) acknowledged this difficulty when he wrote:

'Obtundants have been tried by hundreds of dentists and then faded out of the memory of men. Such has been the fate of every obtundant for sensitive dentine, except for a few now on trial, that have come forward during 70 or more years of clinical practice. But the relief of suffering is an ever-present duty and the search for this very desirable thing should continue.'

This search, however, is beset by problems as other investigators have suggested:

'because of their subjective nature many of the earlier reports on desensitization have little scientific basis and belong in the realm of testimonials.'

(Everett et al. 1966)

'There may well be no single phenomenon in all of science which has occupied so much attention over so much time and yielded so few results as dentin hypersensitivity. Its causes have remained obscure throughout the existence of mankind.'

(Emling 1982)

The problems of methodology and assessment of desensitizing dentifrices has been reviewed in this thesis and it is apparent that despite recent efforts to develop reproducible stimuli more suited to the evaluation of CDS, no single method of assessing CDS may be considered ideal. The absence of suitably objective methods of assessment and the lack of standardized measurement of subjective response following application of stimuli give cause for concern.

The use of low abrasives in dentifrices formulated for the treatment of CDS has been investigated in several clinical studies (**Chapter 3**). While various in vitro studies have indicated that the low abrasive

component, silica, in particular, may partly occlude dentinal tubules (Pashley 1984, Mostafa 1985, Mostafa et al.1986, Absi et al.1989b), results from clinical studies appear to be conflicting. For example, if reduction in sensitivity can be attributed to the silica-based component (Addy et al.1987b), then one would have expected the group using the precipitated silica dentifrice in the 8-week study to have demonstrated significantly greater reduction in sensitivity compared to the diatomaceous earth group. It should be noted, however, that the silica components used in the various clinical studies may have differences in chemical composition, which could account for the differing results observed. The results from the original 8-week study demonstrated that the 2 SCH dentifrices, similar except for their respective abrasive systems, were equally effective in reducing CDS (**Tables 2.5.-2.8.**), and would appear to confirm McFall and Hamrick's (1987) conclusions suggesting that the abrasive component is unlikely to have been responsible for the observed reduction in CDS. These results also highlight the discrepancy that may occur between laboratory and clinical findings, which may not necessarily favour the former.

Problems associated with the methodology used to evaluate the effectiveness of a desensitizing dentifrice have been addressed elsewhere (**section 1.3.**). In the 8 and 20-week studies, the Yeaple probe (quantifiable) and cold air stimuli (semi-quantifiable) were utilised, together with subject assessment of pain using VAS scores. All appeared satisfactory for the measurement of subject response (**Tables 2.5.-2.8. & 3.1.-3.4.**).

Both placebo and associated non-placebo effects have also been reported in studies of this nature (Karlson & Penney 1975, Peden 1977, Addy & Dowell 1983), and while a placebo effect was possible the study was randomised and double-blind and the patients were in no way informed in a manner which would have implied efficacy for either dentifrice.

The inclusion of a placebo group in this study may have enhanced the

findings, but this study concerned primarily the question of abrasivity, and in this context the diatomaceous earth dentifrice was used only as a positive control. Furthermore, a previous study (Minkoff & Axelrod 1987) using a similar range of assessment methods demonstrated that SCH dentifrice produced a significantly greater reduction in sensitivity than placebo.

The results from the 8 and 20-week studies would, however, indicate that the efficacy of SCH dentifrices in reducing CDS was neither immediate nor permanent in its action and like most, if not all, of the desensitizing agents reviewed in this thesis failed to fulfil Grossman's (1935) postulates regarding an ideal desensitizing agent or technique.

To date, despite claims to the contrary, no desensitizing agent or technique appears to completely fulfil these postulates.

Several investigators have suggested that plaque may play a role in the aetiology of CDS (Everett *et al.* 1966, Grant *et al.* 1972, Chasens 1974, Schluger *et al.* 1977, Carranza 1984). Other work indicates that the level of plaque control is not a significant aetiological factor in CDS (Dowell *et al.* 1985), although several assert the importance of good oral hygiene in the management of CDS (Grant *et al.* 1972, Chasens 1974, Schluger *et al.* 1977, Carranza 1984, Hovgaard *et al.* 1988). One of the problems, however, in comparing the effects of oral hygiene on CDS is that a variety of methods have been utilised to record the oral health status of participants. Toto *et al.* (1958) reported that oral hygiene ranged from poor to good, whereas Manochehr-Pour *et al.* (1984) reported that most participants showed an improvement in oral hygiene during the course of the study, although no attempt was made to record plaque. More recent studies (Clark *et al.* 1985, Silverman 1986, Hovgaard *et al.* 1988, Salvato *et al.* 1989, Addy *et al.* 1990b) attempted to measure plaque by partial or whole mouth recording, utilising the Greene and Vermillion (1960) or the Silness and L  e (1964) indices. One of the problems with the Greene and Vermillion Index is that it is difficult to differentiate between plaque and other matter once stained by a

disclosing solution, resulting in an inaccurate assessment of plaque itself. In this study, the Silness and L  e Plaque Index (1964) was used to record (by probe) plaque at six sites on all teeth excluding third molars. Several desensitizing studies (Zinner et al.1977, Gedalia et al.1978, Silverman 1985, Addy et al.1990b) made no attempt to change the oral hygiene practices of participants during the study, whereas Shapiro et al. (1970a,b) and Hovgaard et al. (1988) attempted to carefully controlled hygiene procedures by instruction, reinforced at each visit and corrected if necessary (Shapiro et al.1970a,b). Other investigators (Gedalia et al.1978, Clark et al.1985), however, found even when oral hygiene procedures were not changed prior to inclusion in desensitization dentifrice studies, that there was little significant difference in plaque index between the groups.

In the plaque study, no attempt was made to change the participants' oral hygiene, but all patients received prior oral hygiene instruction and debridement which may account for the relatively low plaque and gingival index scores at the commencement of the study. The slight increase in plaque and gingival scores in the two weeks following baseline readings, and the levelling out of the mean values, may be explained by a slight relapse in oral hygiene following pre-treatment values, and subsequent stabilised maintenance thereafter (Garcia-Godoy et al.1990). It was also observed that no further change in PlI and GI occurred after two weeks. There was no evidence to suggest that any apparent change in the mean plaque and gingival scores reflected an actual change in magnitude. Neither plaque accumulation nor gingival condition significantly changed from baseline levels during the course of the study. The results of this study appear to support the observations of Gedalia et al. (1978) and of Clark et al. (1985) in that there was little or no change between the two groups in plaque scores. Indeed, the plaque effect was identical in both the silica-based (test) and diatomaceous earth (control) groups.

In summary, there was no evidence to suggest that SCH dentifrices increased plaque accumulation, or that the abrasivity of the

desensitizing dentifrice affected the level of plaque.

There is a possibility, however, that the differences observed between the present study and the Addy *et al.* (1990b) study may be due to the different recording techniques used to measure plaque. In the Addy *et al.* study the Greene & Vermillion (1960) Index was used, whereas this study utilised the Silness & L  e (1964) Index. No feasible explanation can be given for this apparent anomaly between the two studies.

The results from the study designed to compare the various methods of evaluating patient subjective response to the test stimuli indicated that both verbal and non verbal techniques were able to quantify the sensory and affective aspects of pain arising from CDS. The choice of word descriptor, however, is important and care should be taken to use words which correspond to the type of pain experienced by the patient (**Chapter 5**). The sequence of application of the various stimuli is also important and has been addressed elsewhere (**section 1.3.**).

In this particular study all methods of assessment demonstrated that patients perceived air stimulation (dental unit syringe) to cause the greatest discomfort and tactile sensation the least, which appears to substantiate the sequence of application as used in the 8 and 20-week studies.

MPQs have been successfully used in various pain studies, although one main criticism is the complexity of the vocabulary. Comparison of recorded scores from patients involved in the 8-week study, demonstrated a very low percentage reproducibility (78/217 [36%]; 70/201 [34.8%] respectively for Gp A & B). This does not suggest a particularly satisfactory relationship between the choice of words over the two visits and may indicate that patients have difficulty in consistently choosing a word to describe CDS.

Other investigators have described the pain arising from CDS as being rapid in onset, sharp in character and short in duration (Tarbet *et al.* 1980, Trowbridge 1991), dull or vague (Stephan 1937, Chasens 1974). In the present study, words most commonly selected to describe pain arising from CDS, were sharp, tender, annoying, stabbing, aching and

nagging.

Thermo-electric devices have been used in the animal model to determine nerve response (Närhi et al.1982) and in humans to assess pre- and post-treatment sensitivity levels (Naylor 1961, Smith & Ash 1964a,b, Kanouse & Ash 1969, Dayton et al.1974, Green et al.1977, Ong 1983, Addy et al.1987b). These devices appear to have the advantage of precise control of temperature in order to provide accurate threshold values following stimulation, although considerable time may be required to set the necessary temperature range (Green et al.1977).

One of the problems with these thermal devices is that they measure temperature at the probe tip and not directly that at the tooth surface, and as such may suffer from a lag between probe and tooth surface temperatures. Consequently changes in temperature must be made slowly in order that a sensitivity threshold is not bypassed (Clark & Troullos 1990).

Discrepancies between set temperatures, as indicated by the digital display, and recorded probe tip temperature may also occur as observed in the in vitro study. This problem may arise for a variety of reasons. The data from the BTP's digital meter is electronically calibrated through electromotive force (EMF) which is generated from the thermocouple in the probe tip, this being the signal data to control the designated temperature. Following initial problems with control of probe temperature, this thermocouple had to be resited adjacent to the Peltier device and not the probe tip, otherwise there would be a delay in monitoring temperature at the thermal generator (Peltier device). The overall effect of such a delay could result in a differential of $\pm 4^{\circ}\text{C}$, which would be unacceptable. The positioning of the thermocouple detector adjacent to the Peltier device resolved the problem, although the effect of this was to allow a slight discrepancy between tip and indicated temperature at the extremes of the temperature range. In the present study the maximum variation between digital display/BTP tip and set temperature values, however, did not exceed a difference of 2.2°C for cold and 2.7°C for hot (**Figs. 6.3.-6.6.**).

This problem may have been exaggerated since the probe tip temperatures were obtained from direct contact of a thermocouple attached to the tip by a silicon sleeve, which was not an integral feature of the BTP, and would not normally be used in the clinical situation. The output of this external meter (Comark Digital thermocouple 5000, Comark UK) can be affected by the localised conditions surrounding the tip thermocouple, including ambient air and contact pressure and may account for the slight, but negligible discrepancies between the digital display and probe tip readings (**Figs. 6.3.-6.6.**) and subsequent lag in time required to achieve the set temperatures in the in vitro situation (**Tables 6.5.-6.7.**).

The results indicated that the digital display temperature reading (**Fig. 6.3.**) was the most accurate and more consistent when compared to the other temperature readings (**Figs. 6.4.-6.6.**), although again these differences were negligible. This would indicate, for all practical purposes, that for any given temperature setting the digital display and BTP tip temperatures were virtually identical. In other words, the set temperature reading as indicated on the digital display would accurately reflect the actual temperature registered at the BTP tip.

There was no variation in the times required to obtain the set temperature values for the BTP, except for the values obtained from 59.9-0°C and 24-0°C settings (**Tables 6.5.-6.7.**).

Overall, the results show a remarkable degree of consistency both in the measurement of temperature and in times required for the BTP to reach these set temperatures.

Previous thermo-electric devices were claimed to have the advantage over other methods of stimulation of precise control of temperature and of providing accurate threshold values. Unfortunately considerable time is required to set the necessary range of temperatures during testing (Green et al. 1977).

The BTP appears to provide a more accurate, reproducible and reliable method of assessing subjective response from cold stimulation than previously reported. In common with other thermal devices it registered

the temperature of the probe tip, and not directly that at the tooth surface, and as such may suffer from a lag between probe and tooth surface temperatures, although in real terms this difference in temperature proved negligible and did not appear to cause any problems in the clinical study.

Considerable care has to be taken when measuring responses to changes in stimulation temperature so that the actual sensitivity threshold is not bypassed (Clark & Troullos 1990). Contrary to the commonly held belief that only pain is registered when a cold stimulus is applied to exposed dentine, patients can differentiate between cold and pain provided changes in temperature are made slowly (Grüsser *et al.* 1982). The Grüsser *et al.* data may, however, be misleading since it was also observed that this relationship was reduced following gingival anaesthesia. The possibility that C-fibres may also have been activated by cold stimulation, however, cannot be ruled out (Jyväsjarvi & Kniffki 1992). Care is also required to prevent the probe tip from coming close to the gingivae where periodontal nerve fibres may be stimulated, this could create difficulties for the patient in determining the exact location of the perceived sensation.

There may also be a problem with placement of a metal tip, even at tooth temperature, on exposed dentine, which may trigger a painful response and consequently preclude further testing. In fact, this was not observed, except in one patient who was highly stressed on the day of testing.

Problems may also arise with inadequate probe contact which can result in the presentation to the tooth of poorly characterised and quantified stimuli (Person *et al.* 1989). For example, as the tip of the BTP was convex in design, it may not have been possible to maintain a uniform contact with the tooth during testing. It would be possible, however, to use a heat sink material to assist heat exchange at the tooth surface, but this was not considered practical in the clinical studies due to the nature and extent of the testing procedures. Criticism has also been made that thermal probes are not representative of the real

life clinical situation (Clark & Troullos 1990), since patients who experience CDS normally complain of cold air or cold liquids and not cold solid objects.

The results from the present clinical studies, however, indicate that the BTP was well tolerated, and perceived to be less traumatic when applied to the tooth surface compared to cold air from a dental unit syringe. Indeed it may be noted that lower temperatures were tolerated by patients when the BTP was applied, which would suggest that the blast component (pressure) from the dental unit syringe provides the major contribution to discomfort perceived by patients following such application of cold, rather than temperature per se. The degree to which the stimulus from a thermoelectric device may be considered mechanical (tactile) in nature as well as thermal still needs to be resolved (Ash 1986). According to Pashley (1990), thermal stimuli should be regarded as hydrodynamic in nature in that they induce fluid movement or pressure changes indirectly rather than by directly stimulating temperature-sensitive receptors.

Other methods of stimulation such as an electric pulp tester are not normally encountered in real life situations. There appears to be a poor correlation between the voltage values obtained with an electrical stimulus and the pain scale values obtained with normally experienced stimuli (e.g., cold) (Närhi et al. 1991). Fear of experiencing an unknown stimulus, and possible discomfort, may influence patient assessment of pain, and in consequence a lower pain threshold may be recorded. Further, stimulation of the pulp on the basis of applied voltage may fail to represent the exact pain threshold, in as much as the stimulating current depends on varying resistance pathways to the pulp or to other adjacent tissues (Ash 1986). The use of constant current stimulators, as in neurophysiology, capable of delivering an exact current regardless of the resistance of the hard tissues of the tooth, has been advocated (Ash 1986, Pashley 1990). Furthermore, because current flow is the critical variable in stimulating nerves, Pashley (1990) considered constant current stimulators essential in the

study of nerve thresholds and sensitivity, although ideal stimulators of this type do not appear to have been used in the assessment of CDS.

The mode and sequence of applying a stimulus which can be varied in intensity is important. Ash (1986) suggested that an increase or decrease in the level of heat or increase in the level of electrical energy should be monotonic rather than delivered in a random order approach. He concluded that while a continuous increase may not be possible, both incremental as well as continuous increases or decreases in stimulus intensity should occur within a standard time frame.

The order of application when more than one kind of stimulus is used is important. Care should be taken to ensure that the first should not distract from the second, nor the second from the third and so on. The least disturbing stimulus should, therefore, be applied first, with the most disturbing used last (Ash 1986, Clark & Troullos 1990). Several investigators have applied either tactile, electrical or heat stimuli prior to the application of cold air on the basis that the former do not appear to elicit a painful response which could affect the latter (Tarbet et al. 1979, 1980, 1982, Minkoff & Axelrod 1987, Orchardson & Collins 1987b, Addy et al. 1987b, Kern et al. 1989, Person et al. 1989). The applied stimulus must be reproducible and behaviour predictable. Without such quantification it is difficult if not impossible to compare the findings of different investigators (Ash 1986). No method of evaluation, however, may be considered reliable when used alone (Addy & Dowell 1983). There is plainly a need to investigate the measurability and reproducibility of these stimuli using methodologies and instrumentation more related to the clinical situation.

Most investigations designed to evaluate the efficacy of desensitizing agents in CDS appear to quantify response by means of criteria which may be described as objective with regard to the method per se, but in reality are subjective with regard to patient response. To some extent, the evaluation of treatment for CDS is difficult regardless of the methodology employed.

The results, however, indicate that the BTP is both consistent and

accurate in the measurement of threshold temperature and appears to provide an objective method of assessing patient response to thermal stimuli. Indeed it could be argued that any differences between probe scores during evaluation were due more to variability in patient perception of stimulus discomfort, rather than to any inaccuracy of the BTP. A similar observation was made by Person *et al.* (1989) during clinical testing of the Temptronic air delivery system.

The original intention of study 1 was to determine the appropriate interstimulus time interval on the basis of tooth temperature, i.e., the time taken for the tooth to return to baseline temperature prior to the next challenge. In retrospect, this was based on a misunderstanding of the basic neurophysiology of the tooth, since it would appear that nerve recovery is not necessarily related to localised surface temperature changes. From the study it would appear that tooth temperature reverts to baseline within 15-20 seconds following hot stimulation and 30 seconds for cold. It was apparent, however, that repeated cold stimulation from cold air (dental unit syringe) and cold (Biomat) with an interstimulus interval of 30 seconds led to desensitization of the tooth and no further response from patients. Although no statistically significant differences were demonstrated between the two instruments, it did appear that cold air produced desensitization sequentially before the BTP. This would appear to be consistent with Brännström's (1960) observation that following prolonged use of evaporative stimuli (e.g., 5 minutes) dentine remained insensitive to further stimulation provided it was kept dry. Närhi *et al.* 1982 observed in the dog model, that with repeated air blasts the nerve responses became weaker and finally disappeared. A similar finding following thermal stimulation was also observed in the cat model by Kollmann & Matthews (1982). It is interesting, therefore, to speculate on what effect thermal stimulation would have on the alteration of nerve response (if any) in humans and whether this would subsequently effect patient responses. Several studies have suggested an interstimulus interval of up to 5 minutes, although no details as how this figure was determined were presented

(Addy *et al.* 1987b, Muzzin & Johnson 1989). Jyväskylä and Kniffki (1987), however, indicated that nerve recovery following thermal stimulation may take up to one hour. It is evident, however, that conditioning of the tooth does occur following repeated thermal stimulation, although the exact effect on nerve response (if any) cannot be established from the present study. For most of the studies a one minute interstimulus interval was incorporated, although study intervals up to and including 5 minutes are reproducible. The difficulty with thermal assessment is the amount of time required for testing, particularly when determination of threshold values is incorporated into the testing procedure.

The results from the study comparing 5°C incremental vs. average threshold values indicated that there was no statistically significant difference between the two sets of values and, therefore, a simple positive/negative response at each temperature level could be incorporated into future testing without affecting accuracy or reproducibility of the elicited response following cold stimulation (BTP). This would also reduce the amount of time required for testing without unnecessarily conditioning the tooth.

The method of presentation of the stimulus (continuous or decremental/incremental) was also determined, and while scores from both methods appeared consistent, it is suggested that there may be a greater problem with tooth conditioning, particularly at the lower temperatures, with the continuous method of presentation. The decremental/incremental method of presentation, therefore, may be more practical. From a practical standpoint the 5°C decremental/incremental approach coupled with a positive/negative patient response to thermal stimulation may constitute a satisfactory method of assessing CDS.

Most non-sensitive teeth did not respond to cold when tested with the BTP. Dayton *et al.* (1974) indicated that sensitive teeth responded between 10 to 20°C, while study 5 indicated a mean of 8.4°C (95% C.I. 5.83 to 10.91).

Hot thresholds were outside the upper limit of the BTP temperature

range and, while the BTP can be adjusted to enable precise measurement of this threshold, testing teeth with temperatures which lie outside this range ($> 60^{\circ}\text{C}$) may induce detrimental changes in the tooth.

Further, patients with CDS generally complain of cold rather than hot stimuli. Consequently, only cold threshold evaluation should be used for CDS assessment on the basis of temperature.

Comparison of BTP and cold air scores obtained over two examination visits (study 5) demonstrated no statistically significant differences and would indicate that the BTP was reproducible in recording accurate temperature thresholds and compares favourably with the report of Person *et al.* (1989) using an air delivery system.

Overall the Biomat thermal probe performed well throughout the studies and provided a more objective method of assessing thermal (cold) sensitivity than cold air, which is not a purely thermal stimulus.

From these studies it would appear that the BTP and the Yeaple probe, together with patient subjective response to these stimuli would provide suitable methodology for the assessment of the efficacy of desensitizing agents in clinical trials.

CONCLUSIONSChapters 2 and 3

1. The results of the randomised double-blind parallel study of 40 patients with CDS over 8 weeks of product use and subsequent evaluation at 20-weeks demonstrated that:
 - i) When assessed by tactile and cold air stimuli, together with patient subjective response, the SCH dentifrices were equally effective and seemed to act to the same degree in relation to time.
 - ii) The response to both dentifrices was evident within 4 weeks of use and the degree of improvement increased during the study.
 - iii) Changing the abrasive component of SCH dentifrices did not significantly increase or decrease the desensitizing activity of the original product.
 - iv) On the basis of the 3 accepted methods of assessment, there was a very slight and not statistically significant change in mean sensitivity levels following the end of the 8-week clinical study.
 - v) Although there was a very slight reversal of the trend demonstrated during the original study, no apparent differences were detected between the silica-based low abrasive and diatomaceous earth groups at the 20-week time point and overall, sensitivity remained significantly lower than at baseline.
 - vi) Reductions in sensitivity achieved by both dentifrices were still evident 3 months after cessation of regular controlled use, and abrasivity did not affect desensitizing activity.
 - vii) SCH dentifrices, however, do not appear to provide immediate

or permanent relief from CDS and, as such, fail to completely fulfil Grossman's (1935) postulates with regard to an ideal desensitizing agent or technique.

Chapter 4

1. The results of the 8-week plaque study do not support the previous conclusions that SCH dentifrices increased plaque accumulation. Neither SCH dentifrices had any clinically significant effect on plaque or gingival condition.

Chapter 5

1. The results of the study evaluating patient subjective response would confirm the conclusions of (Duncan et al. (1989) in that both verbal and non-verbal techniques appear to satisfactorily quantify sensory and affective aspects of pain. The imprecise nature of UVD words, however, provided limited information in terms of accuracy or sensitivity, except at very low levels of discomfort, when assessing pain from CDS.
2. The results of the MPQ study demonstrated that patients were not consistent over the two visits concerning selection of word descriptors, although overall, the most frequently selected words, such as sharp, tender, shooting etc, described the characteristics of pain arising from CDS as reported by others.

Chapter 6

1. The results of the in vitro probe calibration and in vivo clinical studies demonstrated that:
 - i) The BTP was both accurate and consistent in the in vitro measurement of temperature over the range 0°C-59.9°C.
 - ii) At least one to five minutes is required prior to re-application of a cold stimulus by the Biomat Thermal Probe and

dental unit syringe.

- iii) A decremental 5°C one pass positive/negative presentation commencing from 25°C, with a minimum interstimulus interval of one to five minutes, is advisable to prevent desensitization of the tooth during thermal (cold) stimulation with the BTP.
 - iv) Non-sensitive teeth do not normally respond to tactile, cold air (dental unit syringe) or cold (BTP) stimuli, whereas sensitive teeth tend to respond to all these different methods of assessment.
 - v) Comparison of the Biomat Thermal Probe with other methods of assessment, namely cold air from a dental unit syringe, demonstrated that the BTP provided an objective means of assessing patient response following thermal stimulation.
 - vi) The BTP was both accurate and consistent in the measurement of cold threshold stimulation temperatures and therefore, would be useful as a means of objectively assessing patient response to thermal stimuli in clinical studies designed to evaluate the efficacy of desensitizing agents.
- Further clinical studies involving desensitizing agents are, therefore, indicated to compare the BTP with other recognised methods of assessing CDS.

BIBLIOGRAPHY

- ABEL, I. (1958)** Study of hypersensitive teeth and a new therapeutic aid. *Oral Surgery, Oral Medicine, Oral Pathology* 11, 491-495.
- ABSI, E.G., ADAMS, D. & ADDY, M. (1985)** Dentine hypersensitivity. A scanning electronmicroscopic (SEM) study of the effects of mechanical abrasion and dietary compounds on the patency of root surface dentinal tubules. *Journal of Dental Research* 64, 687 (Abstract no. 217).
- ABSI, E.G., ADDY, M. & ADAMS, D. (1987)** Dentine hypersensitivity: A study of the patency of dentinal tubules in sensitive and non-sensitive cervical dentine. *Journal of Clinical Periodontology* 14, 280-284.
- ABSI, E.G., ADDY, M. & ADAMS, D. (1989a)** Dentine hypersensitivity: The development and evaluation of a replica technique to study sensitive and non-sensitive cervical dentine. *Journal of Clinical Periodontology* 16, 190-195.
- ABSI, E.G., ADDY, M. & ADAMS, D. (1989b)** Dentine hypersensitivity. Uptake of various sensitizing toothpastes onto dentine in vitro S.E.M. investigation. *Journal of Dental Research* 68 (Special Issue), 573 (Abstract no. 117).
- ABSI, E.G., ADDY, M. & ADAMS, D. (1992)** Dentine hypersensitivity - the effect of toothbrushing and dietary compounds on dentine in vitro: an SEM study. *Journal of Oral Rehabilitation* 19, 101-110.
- ACCEPTED DENTAL THERAPEUTICS (1979)** Fluoride compounds: Desensitizing action. In *Accepted Dental Therapeutics* 38th edition, American Dental Association, Chicago p 333.
- ADA AD HOC ADVISORY COMMITTEE (1986)** Recommendations for evaluating agents for the reduction of dentinal hypersensitivity. *Endodontics & Dental Traumatology* 2, 172-174.
- ADDY, M. (1990)** Etiology and clinical implications of dentine hypersensitivity. *Dental Clinics of North America* 34, 503-514.
- ADDY, M. (1992)** Clinical aspects of dentine hypersensitivity. *Proceedings of the Finnish Dental Society* 88 (Supplement 1), 23-30.
- ADDY, M., ABSI, E.G. & ADAMS, D. (1987a)** Dentine hypersensitivity: The

effects in vitro of acids and dietary substances on root planed and burred dentine. Journal of Clinical Periodontology 14, 274-279.

ADDY, M. & DOWELL, P. (1983) Dentine hypersensitivity - A review. Clinical and in vitro evaluation of treatment agents. Journal of Clinical Periodontology 10, 351-363.

ADDY, M., LOYN, T. & ADAMS, D. (1990a) The effects in vitro of some mouthwash products on the dentine smear layer. Journal of Dental Research 69 (Special Issue), 966 (Abstract no. 89).

ADDY, M., LOYN, T. & ADAMS, D. (1991) Dentine hypersensitivity - effects of some proprietary mouthwashes on the dentine smear layer. A SEM study. Journal of Dentistry 19, 148-152.

ADDY, M. & MORGAN, T. (1982) Scanning electron microscope (SEM) and electrical resistance changes in dentine treated with toothpastes. Journal of Dental Research 61, 547 (Abstract no. 102).

ADDY, M. & MOSTAFA, P. (1988) Dentine hypersensitivity: I. Effects produced by the uptake in vitro of metal ions, fluoride and formaldehyde onto dentine. Journal of Oral Rehabilitation 15, 575-585.

ADDY, M. & MOSTAFA, P. (1989) Dentine hypersensitivity. II. Effects produced by the uptake in vitro of toothpastes onto dentine. Journal of Oral Rehabilitation 16, 35-48.

ADDY, M., MOSTAFA, P., ABSI, E.G. & ADAMS, D. (1985) Cervical dentin hypersensitivity. Etiology and management with particular reference to dentifrices. In: Rowe, N.H. (ed): Proceedings of Symposium on Hypersensitive Dentin. Origin and Management, University of Michigan, Pp 43-64.

ADDY, M., MOSTAFA, P. & NEWCOMBE, R. (1987b) Dentine hypersensitivity. A comparison of five toothpastes used during a 6-week treatment period. British Dental Journal 163, 45-51.

ADDY, M., MOSTAFA, P. & NEWCOMBE, R.G. (1987c) Dentine hypersensitivity: the distribution of recession, sensitivity and plaque. Journal of Dentistry 15, 242-248.

ADDY, M., MOSTAFA, P. & NEWCOMBE, R.G. (1990b) Effect on plaque of five toothpastes used in the treatment of dentin hypersensitivity. Clinical

Preventive Dentistry 12, 28-33.

ADRIAN, J.C. (1977) Pulpal effects of neodymium laser. A preliminary report. Oral Surgery, Oral Medicine, Oral Pathology 44, 301-305.

ADRIAN, J.C., BERNIER, J.L. & SPRAGUE, W.G. (1971) Laser and the dental pulp. Journal of the American Dental Association 83, 113-117.

ANDERSON, D.J. (1963) Chemical and osmotic excitant of pain in human dentine. In. Sensory Mechanisms in Dentine, ANDERSON, D.J. (ed), Pergamon Press, Oxford Pp 88-89.

ANDERSON, D.J. (1968) The pulp as a sensory organ. In. Biology of the Dental Pulp Organ, Finn, S.B. (ed) Birmingham, Alabama, University of Alabama Press Pp 273-280.

ANDERSON, D.J. (1972) Human and animal studies on sensory mechanisms in teeth. Journal of Dental Research 22, 33-38.

ANDERSON, D.J., CURWEN, M.P. & HOWARD, L.V. (1958). The sensitivity of human dentin. Journal of Dental Research 37, 669-677.

ANDERSON, D.J., HANNAM, A.G. & MATTHEWS, B. (1970) Sensory mechanisms in mammalian teeth and their supporting structures. Physiological Reviews 50, 171-195.

ANDERSON, D.J. & MATTHEWS, B. (1966) An investigation into the reputed desensitising effect of applying silver nitrate and strontium chloride to human dentine. Archives of Oral Biology 11, 1129-1135.

ANDERSON, D.J. & MATTHEWS, B. (1967) Osmotic stimulation of human dentine and the distribution of dental pain thresholds. Archives of Oral Biology 12, 417-426.

ANDERSON, D.J., MATTHEWS, B. & GORRETTA, C. (1967a) Fluid flow through human dentine. Archives of Oral Biology 12, 209-216.

ANDERSON, D.J., MATTHEWS, B. & SHELTON, L.E. (1967b) Variations in the sensitivity of osmotic stimulation of human dentine. Archives of Oral Biology 12, 43-47.

ANDERSON, D.J. & NAYLOR, M.N. (1962) Chemical excitants of pain in human dentine and dental pulp. Archives of Oral Biology 7, 413-415.

ANDERSON, D.J. & RONNING, G.A. (1962) Osmotic excitants of pain in human dentine. Archives of Oral Biology 7, 513-523.

- ARAUJO, P.A. de. & ASMUSSEN, E. (1989) Aluminium oxalate/glycine solutions as pretreatment in the Gluma bonding system. *Scandinavian Journal of Dental Research* 97, 552-558.
- ARWILL, T. (1967) Studies on the ultrastructure of dental tissues. II. The predentine-pulpal border zone. *Odontologisk Revy* 18, 191-208.
- ARWILL, T. (1968) The ultrastructure of the pulpo-dentinal border zone. In: *Dentine and Pulp. Their Structure and Functions*. Symons, N.B.B. (ed) E. & S. Livingstone Limited, Edinburgh & London Pp 147-167.
- ARWILL, T., EDWALL, L., LILJA, J. & SVENSON, S.E. (1973) Ultrastructure of nerves in the dentinal-pulp border zone after sensory and autonomic nerve transection in the cat. *Acta Odontologica Scandinavica* 31, 273-281.
- ASH, M.M. (1986) Quantification of stimuli. *Endodontics & Dental Traumatology* 2, 153-156.
- ASMUSSEN, E. & BOWEN, R.L. (1987) Effect of acidic pretreatment on adhesion to dentin mediated by Gluma. *Journal of Dental Research* 66, 1386-1388.
- AUBIN, J.E. (1985) New immunological approaches to studying the odontoblast. *Journal of Dental Research* 54, 515-522.
- AVERY, J.K. (1971) Structural elements of the young normal pulp. *Oral Surgery, Oral Medicine, Oral Pathology* 32, 113-125.
- AVERY, J.K. (1974) Anatomical considerations in the mechanisms of pain and sensitivity in the teeth and supporting structures. In: Chasens, A.I. & Kaslick, R.S. (eds) *Workshop on Mechanisms of Pain and Sensitivity in the Teeth and Supporting tissues*, Fairleigh Dickinson University, School of Medicine & American Academy of Oral Medicine, 12-13 November Pp 16-23.
- AVERY, J.K. & RAPP, R. (1959) An investigation of the mechanism of neural impulse transmission in human teeth. *Oral Surgery, Oral Medicine, Oral Pathology* 12, 190-198.
- BARBAKOW, F., GIUDICI, S. & LUTZ, F. (1992) Effects of precipitate free SnF₂-Amine Fluoride 297 gels on dentin dissolution & fluoride retention. *Journal of Dental Research* 71, 194 (Abstract no. 706).

- BARBAKOW, F., LUTZ, F. & SENER, B. (1986) Fluoride retention of enamel brushed in vitro with nicomethanol-hydrofluoride. Journal of Dental Research 65, 184 (Abstract no. 134).
- BARBAKOW, F., LUTZ, T. & IMFELD, L. (1987a) Abrasives in dentifrices and prophylaxis pastes. Quintessence International 18, 17-22.
- BARBAKOW, F., LUTZ, T. & IMFELD, L. (1987b) A review of methods to determine the relative abrasion of dentifrices and prophylaxis pastes. Quintessence International 18, 23-28.
- BARBAKOW, F., LUTZ, T. & IMFELD, L. (1987c) Relative dentin abrasion by dentifrices and prophylaxis pastes. Implications for clinicians, manufacturers and patients. Quintessence International 18, 29-34.
- BASTOS, P., TEIXEIRA, L. & LEINFELDER, K. (1991) Three-year clinical evaluation of Scotchbond 2 as a dentinal adhesive. Journal of Dental Research 70, 395 (Abstract no. 1031).
- BEASLEY, W.L. & HOLLAND, G.R. (1978) A quantitative analysis of the innervation of the pulp of the cat's canine tooth. Journal of Compendium of Neurophysiology 178, 487-494.
- BENDER, I.B. (1978) Pulp Biology Conference. A discussion. Journal of Endodontics 4, 37-52.
- BERGENHOLTZ, G. (1977) Effect of bacterial products on the inflammatory reactions in the dental pulp. Scandinavian Journal of Dental Research 85, 122-129.
- BERGENHOLTZ, G., COX, C.F., LOESCHE, W.J. & SYED, S.A. (1982) Bacterial leakage around restorations. Its effects on the dental pulp. Journal of Oral Pathology 11, 439-450.
- BERGENHOLTZ, G. & LINDHE, J. (1975) Effect of soluble plaque factors on the inflammatory reactions in the dental pulp. Scandinavian Journal of Dental Research 85, 153-158.
- BERGGREN, G. & BRÄNNSTRÖM, M. (1965) The rate of flow in dentinal tubules due to capillary action. Journal of Dental Research 44, 408-415.
- BERMAN, L.H. (1985) Dentinal sensation and hypersensitivity. A Review of mechanisms and treatment alternatives. Journal of Periodontology 56,

216-222.

BERNICK, S. (1948) Innervation of the human tooth. *Anatomical Record* 101, 81-108.

BERNICK, S. (1968) Innervation of the teeth. In. *Biology of the Dental Pulp Organ*, Finn, S.B. (ed), Birmingham, Alabama, University of Alabama Press Pp 285-302.

BHATIA, H.L. (1953) Use of sodium silicofluoride as a desensitizing agent for exposed sensitive cementum and cervical dentin. *Bur* 54, 4-6.

BILOTTO, G., MARKOWITZ, K. & KIM, S. (1986) Intradental nerve activity in response to ionic and non ionic solutions. *Journal of Dental Research* 65, 210 (Abstract no. 368).

BILOTTO, G., MARKOWITZ, K. & KIM, S. (1987) Experimental procedures to test the efficacy of chemical agents on intradental nerve activity. *Journal of Endodontics* 13, 458-465.

BILOTTO, G., MARKOWITZ, K. & KIM, S. (1988) Effects of ionic and non-ionic solutions on intradental nerve activity. *Pain* 32, 231-238.

BLACK, G.V. (1908) The pathology of the hard tissues of the teeth. In. *A Work on Operative Dentistry in Two Volumes. Volume 1*, Chicago Medico-Dental Publishing Co. Pp 149-150.

BLITZER, B. (1967) A consideration of the possible causes of dental hypersensitivity. Treatment by a strontium-ion dentifrice. *Periodontics* 5, 318-321.

BLONG, M.A., VOLDING, B., THRASH, W.J. & JONES, D.L. (1985) Effects of a gel containing 0.4 percent stannous fluoride on dentinal hypersensitivity. *Dental Hygiene* 59, 489-492.

BLOSSER, R.L. & BOWEN, R.L. (1986) Adhesive bonding to composites; Ferric oxalate and nitric acid concentrations. *Journal of Dental Research* 65, 315 (Abstract no. 1311).

BLOSSER, R.L. & BOWEN, R.L. (1988) Effects of purified ferric oxalate/nitric acid solutions as a pretreatment for the NTG-GMA and PMDA bonding system. *Dental Materials* 4, 225-231.

BLOSSER, R.L., RUPP, N.W., STANLEY, H.R. & BOWEN, R.L. (1988) Pulpal response to two new dentin and enamel bonding systems. *Journal of*

Dental Research 67, 134 (Abstract no. 174).

BLUNDEN, R., HOWE, S., DOWELL, P, ADDY, M., ADAMS, D. & MCCARTHY, J. (1981) The effects of compounds used clinically in the management of dentine hypersensitivity on some physical properties of dentine.

Journal of Dental Research 60, 1172 (Abstract no. 130).

BOLDEN, T. E., VOLPE, A.R. & KING, W.J. (1968) The desensitizing effect of a sodium monofluorophosphate dentifrice. Periodontics 6, 112-114.

BOWEN, R.L. (1986) Substitution of aluminum oxalate for ferric oxalate in adhesive bonding. Journal of Dental Research 65, 239 (Abstract no. 627).

BOWEN, R.L. & COBB, E.N. (1983) A method for bonding to dentin and enamel. Journal of the American Dental Association 107, 734-736.

BOWEN, R.L., COBB, E.N. & RAPSON, J.E. (1982) Adhesive bonding of various materials to hard tooth tissues. Improvement in bond strength to dentin. Journal of Dental Research 61, 1070-1076.

BOWEN, R.L., RUPP, N.W., EICHMILLER, F.C. & STANLEY, H.R. (1987a) Clinical trial of a material adhesive to dentin and enamel. Journal of Dental Research 67, 284 (Abstract no. 1373).

BOWEN, R.L., TUNG, M.S., BLOSSER, R.L. & ASMUSSEN, E. (1987b) Dentine and enamel bonding agents. International Dental Journal 37, 158-161.

BOWERS, G.M. & ELLIOT, J.R. (1964) Topical use of prednisolone in periodontics. Journal of Periodontology 35, 486-488.

BRADY, B.H.G., NAPPER, D.H. & SYMTHE, B.M. (1966) Dissolution kinetics of hydroxyapatite. Nature 212, 77-78.

BRADY, B.H.G., NAPPER, D.H. & SYMTHE, B.M. (1968) Effect of additives on the rate of dissolution of hydroxyapatite in unstirred acid buffers. Journal of Dental Research 47, 603-612.

BRÄNNSTRÖM, M. (1960a) Dentinal and pulpal response - 1. Application of reduced pressure to exposed dentine. An experimental study. Acta Odontologica Scandinavica 18, 1-15.

BRÄNNSTRÖM, M. (1960b) Dentinal and pulpal response - 2. Application of an air stream to exposed dentine. An experimental study. Acta Odontologica Scandinavica 18, 17-28.

- BRÄNNSTRÖM, M. (1962) The elicitation of pain in human dentine and pulp by chemical stimuli. Archives of Oral Biology 7, 59-62.
- BRÄNNSTRÖM, M. (1963a) A hydrodynamic mechanism in the transmission of pain producing stimuli through the dentine. In: Sensory Mechanisms in Dentine, Anderson, D.J. (ed) Oxford Pergammon Press Pp 73-79.
- BRÄNNSTRÖM, M. (1963b) Dentin sensitivity and aspiration of odontoblasts. Journal of American Dental Association 66, 366-370.
- BRÄNNSTRÖM, M. (1966) Sensitivity of dentine. Oral Surgery, Oral Medicine, Oral Pathology 21, 517-526.
- BRÄNNSTRÖM, M. (1968) Dentinal and Pulpal Reaction. Physio-pathological aspects of dentinal and pulpal response to irritants. In. Dentine and Pulp. Their Structures and Functions. Symons, N.B.B (ed) E. & S. Livingstone Limited, Edinburgh & London Pp 231-246.
- BRÄNNSTRÖM, M. (1979) The transmission and control of dentinal pain. In. Mechanisms and Control of Pain. Grossman, L.T. (ed) Masson Publications, New York.
- BRÄNNSTRÖM, M. (1981) Dentin and Pulp in Restorative Dentistry. Dental Therapeutics AB, Sweden Nacka Pp 20-24 & 87-88.
- BRÄNNSTRÖM, M. & ÅSTRÖM, A. (1964) A study on the mechanism of pain elicited from the dentin. Journal of Dental Research 43, 619-625.
- BRÄNNSTRÖM, M. & ÅSTRÖM, A. (1972) The hydrodynamics of the dentine; its possible relationship to dentinal pain. International Dental Journal 22, 219-227.
- BRÄNNSTRÖM, M. & GARBEROGLIO, R. (1972) The dentinal tubules and the odontoblastic processes. Acta Odontologica Scandinavica 30, 291-311.
- BRÄNNSTRÖM, M. & GABEROGLIO, R. (1980) Occlusion of dentinal tubules under superficial attrited dentin. Swedish Dental Journal 4, 87-91.
- BRÄNNSTRÖM, M., ISACSSON, G. & JOHNSON, G. (1976) The effect of calcium hydroxide and fluorides on human dentine. Acta Odontologica Scandinavica 34, 59-67.
- BRÄNNSTRÖM, M. & JOHNSON, G. (1970) Movements of the dentine and pulp fluids as application of thermal stimuli: An in vitro study. Acta Odontologica Scandinavica 28, 59-70.

- BRÄNNSTRÖM, M. & JOHNSON, G. (1978) The sensory mechanism in human dentine as revealed by evaporation and mechanical removal of dentine. *Journal of Dental Research* 57, 49-53.
- BRÄNNSTRÖM, M., JOHNSON, G. & NORDENVALL, K.J. (1979) Transmission and control of dentinal pain: resin impregnation for the desensitization of dentin. *Journal of the American Dental Association* 99, 612-618.
- BRÄNNSTRÖM, M., LINDÉN, L.A. & ÅSTRÖM, A. (1967) The hydrodynamics of the dental tubule and of pulp fluid. A discussion of its significance in relation to dentinal sensitivity. *Caries Research* 1, 310-317.
- BRODIN, P. & ROED, A. (1984) Effects of eugenol on rat phrenic nerve and phrenic nerve-diaphragm preparations. *Archives of Oral Biology* 29, 611-615.
- BROUGH, K.M., ANDERSON, D.M., LOVE, J. & OVERMAN, P.R. (1985) The effectiveness of iontophoresis in reducing dentin hypersensitivity. *Journal of the American Dental Association* 111, 761-765.
- BUCKLEY, L.A. (1981) The relationship between malocclusion, gingival inflammation, plaque and calculus. *Journal of Periodontology* 52, 35-40.
- BUELTSMANN, K.W., KARLSSON, U.L. & EDIE, J. (1972) Quantitative ultrastructure of intradental nerve fibres in marmosets. *Archives of Oral Biology* 17, 645- 660.
- BURMAN, L.R. (1963) Desensitizing agents and their role in clinical dentistry. *Journal of American Geriatrics Society* 11, 147-155.
- BURMAN, L.R. & GOLDSTEIN, A. (1961) Evaluation of a desensitizing agent by response to a controlled external stimulus. *Journal of Periodontology* 32, 257-260.
- BYERS, M.R. (1979) Large and Small Trigeminal Nerve Endings and Their Association with Odontoblasts in Rat Molar Dentin and Pulp. In. *Advances in Pain Research Therapy*, Bonica, J.J., Liebeskind, J.C. & Albe-Fessard, D.G. (eds) New York, Raven press Vol 3, Pp 265-270.
- BYERS, M.R. (1984) Dental sensory receptors. *International Review Neurobiology* 25, 39-94.
- BYERS, M.R. (1992) Effects of inflammation on dental sensory nerves and vice versa. *Proceedings of the Finnish Dental Society (Supplement 1)*,

499-506.

BYERS, M.R. & KISH, S.J. (1976) Delineation of somatic nerve endings in rat teeth by radioautography of axon-transported protein. *Journal of Dental Research* 55, 419-425.

BYERS, M.R. & MATTHEWS, B. (1981) Autoradiographic demonstration of ipsilateral and contralateral sensory nerve endings in cat dentin, pulp and periodontium. *Anatomical Record* 201, 249-260.

BYERS, M.R., NÄRHI, M.V. & DONG, W.K. (1987a) Sensory innervation of pulp and dentin in adult dog teeth as demonstrated by autoradiography. *Anatomical Record* 218, 207-215.

BYERS, M.R., NÄRHI, M.V. & DONG, W.K. (1987b) Response of dental sensory receptors to injury of dentin and pulp. In: *Effects of Injury on Trigeminal and Spinal Somatosensory systems*. Pubols, L.M. & Sessle, B.J. (eds), A.R. Liss, Inc., New York Pp 3-10.

BYERS, M.R., NÄRHI, M.V.O. & MECIFI, K.B. (1988) Acute and chronic reactions of dental sensory nerve fibers to cavities and desiccation in rat molars. *Anatomical Record* 221, 872-883.

BYERS, M.R. & TAYLOR, P.E. (1990) Reactions of dental sensory innervation to injury and inflammation. In: *Dynamic aspects of Dental Pulp, Molecular Biology, Pharmacology and Pathophysiology*. Inoki, R., Kudo, T. & Olgart, L.M. (eds) Chapman & Hall, London Pp 425-444.

CADDEN, S.W., LISNEY, S.J.W. & MATTHEWS, B. (1982) A β Fibre innervation of tooth-pulp in the cat, with a discussion of the functions of nerves supplying tooth-pulp. In: *Anatomical, Physiological & Pharmacological Aspects of Trigeminal Pain*. Matthews, B. & Hill, R.G. (eds) Excerpta Medica, Amsterdam Pp 41-49.

CARLO, G.T., CIANCIO, S.G. & SEYREK, S.K. (1982) An evaluation of iontophoretic application of fluoride for tooth desensitization. *Journal of the American Dental Association* 105, 452-454.

CARRANZA, F.A. (1979) *Glickman's Clinical Periodontology*; 5th ed. Philadelphia, Saunders Pp 100-105.

CARRANZA, F. A. (1984) In: *Glickman's Clinical Periodontology* 6th Edition W.B. Saunders Company, Philadelphia p 386.

- CARRASCO, P.H. (1971)** Strontium chloride toothpaste. Effectiveness as related to duration of use. *Pharmacology and Therapeutic Dentistry* 1, 209-215.
- CHAPMAN, C.R. CASEY, K.L., DUBNER, R., FOLEY, K.M., GRACELY, R.H. & READING, A.E. (1985)** Pain measurement. A overview. *Pain* 22, 1-31.
- CHASENS, A.I. (1974)** The management of pain and sensitivity. In: Chasens, A.I. & Kaslick, R.S. (eds) *Workshop on Mechanisms of Pain and Sensitivity in the Teeth and Supporting Tissues*, Fairleigh Dickinson University, School of Medicine & American Academy of Oral Medicine, 12-13 November Pp 12-15.
- CHESTERS, R., KAUFMAN, H.W., WOLFF, M.S., HUNTINGTON, E. & KLEINBERG, I. (1992)** Use of multiple sensitive measurements and logit statistical analysis to assess the effectiveness of a potassium-citrate-containing dentifrice in reducing dentinal hypersensitivity. *Journal of Clinical Periodontology* 19, 256-261.
- CHOHAYEB, A.A., BOWEN, R.L., RUPP, N.W. & ADRIAN, J. (1985)** Pulpal response to a new dentin and enamel system. *Journal of Dental Research* 64, 222 (Abstract no. 428).
- CLARK, D.C. (1982)** A review on fluoride varnishes. An alternative topical fluoride treatment. *Community Dental Oral Epidemiology* 10, 117-123.
- CLARK, D.C., AL-JOBURI, W. & CHAN, E.C.S. (1987)** The efficacy of a new dentifrice in treating dentin sensitivity. Effects of sodium citrate and sodium fluoride as active ingredients. *Journal of Periodontal Research* 22, 89-93.
- CLARK, D.C., HANLEY, J.A., GEOGHEGAN, S. & VINET, D. (1985)** The effectiveness of a fluoride varnish and a desensitizing toothpaste in treating dentinal sensitivity. *Journal of Periodontal Research* 20, 212-219.
- CLARK, D.C., WOO, G., SILVER, J.G., SWEET, D. & GRISDALE, J.C. (1990a)** The influence of frequent ingestion of acids in the diet on treatment of dentin sensitivity. *Journal of the Canadian Dental Association* 56, 1101-1103.

- CLARK, D.C., ITO, S., WOO, G., DUGDALE, K. & TONZETICH, (1990b) Effects of remineralizing mouthrinse on dentin sensitivity. *Journal of Dental Research* 69, 261 (Abstract no. 1218).
- CLARK, G.E. & TROULLOS, E.S. (1990) Designing hypersensitivity clinical studies. *Dental Clinics of North America* 34, 531-544.
- CLEMENT, A.J. (1947) Sodium fluoride paste as a desensitising agent for hypersensitive dentine. *British Dental Journal* 82, 168.
- COFFREY, C.T., INGRAM, M.J. & BJORNDAAL, A. (1970) Analysis of human dentinal fluid. *Oral Surgery, Oral Medicine, Oral Pathology* 30, 835-837.
- COHEN, A. (1961) Preliminary study of the effects of a strontium chloride dentifrice for the control of hypersensitive teeth. *Oral Surgery, Oral Medicine, Oral Pathology* 14, 1046-1052.
- COHEN, J. (1960) A coefficient of agreement for nominal scales. *Educational Psychology Measurement* 20, 37-46.
- COLANERI, J.N. (1952) A simple treatment of hypersensitive cervical dentine. *Oral Surgery, Oral Medicine, Oral Pathology* 5, 276-278.
- COLLAERT, B., DE BRUYN, H., SÖDERHOLM, G. & BRATTHALL, G.T. (1991) Evaluation of a fluoride varnish for dentine sensitivity treatment. *Journal of Dental Research* 70, 83 (Abstract no. 102).
- COLLAERT, B. & FISCHER, C. (1991) Dentine hypersensitivity. *Endodontics & Dental Traumatology* 7, 145-152.
- COLLINS, J.F., GINGOLD, J., STANLEY, H. & SIMRING, M. (1984) Reducing dentinal hypersensitivity with strontium chloride and potassium nitrate. *General Dentistry* 32, 40-43.
- COLLINS, W., ORCHARDSON, R. & GILMOUR, H. (1990) Pilot study of a desensitising agent for dentine. *Journal of Dental Research* 69, 988 (Abstract no. 267).
- COLLINS, J.F. & PERKINS, L. (1984) Clinical evaluation of the effectiveness of three dentifrices in relieving dentin sensitivity. *Journal of Periodontology* 55, 720-725.
- COOLEY, R.L. & SANDOVAL, V.A. (1989) Effectiveness of potassium oxalate treatment on dentin sensitivity. *General Dentistry* July/August, 330-

333.

COPELAND, J.S. (1985) Simplified remedy for root sensitivity. *Northwest Dentistry* 64, 13-14.

CORPRON, R.E. & AVERY, J.K. (1973) The ultrastructure of intradental nerves in developing mouse molars. *Anatomical Record* 175, 585-591.

COUNCIL ON DENTAL THERAPEUTICS. (1985) Acceptance of Sensodyne toothpaste for sensitive teeth. *Journal of the American Dental Association* 110, 394.

COUNCIL ON DENTAL THERAPEUTICS. (1986) ADA Ad hoc Advisory Committee: Recommendations for evaluating agents for the reduction of dentinal hypersensitivity. *Journal of the American Dental Association* 112, 709.

CRAIG, G.C. (1973) Some observations on the use of a calcium sucrose phosphate-calcium orthophosphate complex as a desensitising agent. *Australian Dental Journal* 18, 328-330.

CROSA, M. FERREYRA, S. CROSA, D., INDOVINO, J. & OLIVERA, P. (1991) Laser radiation: Effects on radicular dentine. *Journal of Dental Research*. 70, 640 (Abstract no. 35).

CUENIN, M.F., SCHEIDT, M.J., O'NEAL, R.B., STRONG, S.L., PASHLEY, D.H., HORNER, J.A. & VAN DYKE, T.E. (1991) An *in vivo* study of dentin sensitivity. The relation of dentin sensitivity and the patency of dentin tubules. *Journal of Periodontology* 62, 668-673.

DACHI, S.F. & STIGERS, R.W. (1967) Reduction of pulpal inflammation and thermal sensitivity in amalgam-restored teeth treated with copal varnish. *Journal of the American Dental Association* 74, 1281-1285.

DAHL, E. & MJÖR, I.A. (1973) The structure and distribution of nerves in the pulp-dentine organ. *Acta Odontologica Scandinavica* 31, 349-356.

DAVIS, W.B. & WINTER, P.J. (1980) The effect of abrasion on enamel and dentine after exposure to dietary acid. *British Dental Journal* 148, 253-256.

DAYTON, R.E., DEMARCO, T.J. & SWEDLOW, D. (1974) Treatment of hypersensitive root surfaces with dental adhesive materials. *Journal of Periodontology* 45, 873-878.

DEDERICH, D., ZAKARIASEN, K., TULIP, J., REEH, E. & MORELA, D. (1984)

- SEM analysis of canal-wall dentin following Nd-YAG laser irradiation. *Journal of Dental Research* 63, 171 (Abstract no. 8).
- DESAUTELS, P. & LABRÈCHE, H. (1988)** Abrasion relative des dentifrices - un dentifrice pour chacun. *Journal Dentaire Digest du Québec* 25, 65-74.
- DEVANEY, J.A. (1982)** Letters to the Editor. Toothpaste component. *Journal of the American Dental Association*, 105, 608.
- DOWELL, P. & ADDY, M. (1983)** Dentine hypersensitivity. A review. Aetiology, symptoms and theories of pain production. *Journal of Clinical Periodontology* 10, 341-350.
- DOWELL, P. & ADDY, M. (1984)** Dentine hypersensitivity. A quantitative comparison of the uptake of metal salts and fluoride by dentine and hydroxyapatite. *Journal of Periodontal Research* 19, 530-539.
- DOWELL, P., ADDY, M. & DUMMER, P. (1985)** Dentine hypersensitivity. Aetiology, differential diagnosis and management. *British Dental Journal* 158, 92-96.
- DOWNIE, W.W., LEATHAM, P.A., RHIND, V.M., WRIGHT, V., BRANCO, J.A. & ANDERSON, J.A. (1978)** Studies with pain rating scales. *Annals of the Rheumatic Diseases* 37, 378-381.
- DRIESSENS, F., VERBEECK, R. & ROTGANS, J. (1991)** Aluminium oxyhydroxide in toothpastes and its possible risk. *Journal of Dental Research* 70, 754 (Abstract no. 118).
- DUKE, E.S., ROBBINS, J.W., HOWELL, M.L. & SUMMITT, J.B. (1991a)** Clinical evaluation of a dentin adhesive system in cervical abrasions. *Journal of Dental Research* 70, 395 (Abstract no. 1032).
- DUKE, E.S., ROBBINS, J.W. & SYNDER, D.E. (1991b)** Dentin adhesive evaluation for restoring cervical abrasion lesions. *Journal of Dental Research* 70, 457 (Abstract no. 1529).
- DUMSHA, T.C. & BECKERMAN, T. (1986)** Pulp response to dentin bonding in miniature swine. *Dental Materials* 2, 156-158.
- DUNCAN, G.H., BUSHNELL, M.C. & LAVIGNE G.J. (1989)** Comparison of verbal and visual analogue scales for measuring the intensity and unpleasantness of experimental pain. *Pain* 37, 295-303.
- EDWALL, L. & OLGART, L. (1977)** A new technique for recording

intradental nerve activity in man. *Pain* 3, 121-126.

EDWALL, L. & SCOTT, D. (1971) Influence of changes in microcirculation on the excitability of the sensory unit in the tooth of the cat. *Acta Physiologica Scandinavica* 82, 555-566.

EKOWSKI, C., HRUBES, V., JOYCE, C.R.B., FLOOD, M.K., MASON, R.M. & ZUTSHI, D.W. (1972) An experimental study of two methodolgical problems in clinical evaluation. Different types of scales and the availability of patients' previous judgments. *Psychopharmacology (Supplement)* 26, 70.

ELLINGSEN, J.E. & RÖLLA, G. (1987) Treatment of dentin with stannous fluoride. SEM and electron microprobe study. *Scandinavian Journal of Dental Research* 15, 281-286.

EMLING, R.C. (1982) Historical overview of causes and treatment of dentinal hypersensitivity. *Compendium of Continuing Education in Dentistry Supplement* 3, 92-94.

ENGSTRÖM, H. & ÖHMAN, A. (1960) Studies on the innervation of the human teeth. *Journal of Dental Research* 39, 799-809.

ERHLICH, J., HOCHMAN, H., GEDALIA, I. & TAL M. (1975) Residual fluoride concentrations and scanning electron microscopic examination of root surfaces of human teeth after topical application of fluoride *in vivo*. *Journal of Dental Research* 54, 897-900.

ERLANGER, J. & GASSER, H.S. (1937) *Electrical Signs of Nervous Activity*. University of Pennsylvania press, Philadelphia, 2nd edition Pp 34-78.

EVERETT, F.G. (1964) Desensitization of hypersensitive exposed root surfaces. *Dental Clinics of North America* March Pp 221-230.

EVERETT, F.G., HALL, W.B. & PHATAK, N.M. (1966) Treatment of hypersensitive dentin. *Journal of Oral Therapeutics & Pharmacology* 2, 300-310.

FARMER, J.B. & COX, C.F. (1990) Pulp response following treatment of dentin with various sealing agents. *Journal of Dental Research* 69, 300 (Abstract no. 1535).

FEARNHEAD, R.W. (1957) Histological evidence for the innervation of

human dentine. *Journal of Anatomy* 19, 267-277.

FEARNHEAD, R.W. (1961) The neurohistology of human dentine. *Proceedings of Royal Society of Medicine* 54, 877-884.

FEARNHEAD, R.W. (1963) The histological demonstration of nerve fibres in human dentine. In. *Sensory Mechanisms in Dentine*, Anderson, D.J. (ed) Oxford, Pergamon Press Pp 15-24.

FEARNHEAD, R.W. (1967) Innervation of dental tissues. In. *Structures and Chemical Organisation of Teeth*, Miles, A.E.W. (ed) Academic Press, New York Vol 1 Pp 247-281.

FEINMANN, C. (1984) Facial pain. Psychiatric aspects. *Dental Update*, August 393-400.

FELTON, D.A., BERGENHOLTZ, G. & KANOY, B.E. (1991) Evaluation of the desensitizing effect of Gluma dentin bond on teeth prepared for complete-coverage restorations. *International Journal of Prosthodontics* 4, 292-298.

FISH, E.W. (1927) The circulation of lymph in dentin and enamel. *Journal of the American Dental Association* 14, 804-817.

FISCHER, C., FISCHER, R.G. & WENNBERG, A. (1992) Prevalence and distribution of cervical dentinal hypersensitivity in a population in Rio de Janeiro, Brazil. *Journal of Dentistry* 20, 272-276.

FITZGERALD, G. (1956) A clinical evaluation of a new agent for the relief of hypersensitive dentine. *Dental Digest* 62, 494-497.

FLYNN, J., GALLOWAY, R. & ORCHARDSON, R. (1985) The incidence of 'hypersensitive' teeth in the West of Scotland. *Journal of Dentistry* 13, 230-236.

FORREST, J.O. (1963) A clinical assessment of three desensitising toothpastes containing formalin. *British Dental Journal* 114, 103-106.

FRANK, R.M. (1963) Dentinal sclerosis and ultrastructural basis of dentinal sensitivity. *Journal de Paradontologie* 23, 11-16.

FRANK, R.M. (1968a) Ultrastructural relationship between the odontoblast, its process and the nerve fibre. In *Dentine and Pulp. Their Structures and Functions*. Symons, N.B.B. (ed) E. & S. Livingstone Ltd., Edinburgh & London Pp 115-145.

- FRANK, R.M. (1968b)** Attachment sites between the odontoblast process and the intra-dental nerve fibre. *Archives of Oral Biology* 13, 833-834.
- FRANK, R.M. & STEUER, P. (1988)** Transmission electron microscopy of the human odontoblast process in peripheral root dentine. *Archives of Oral Biology* 33, 91-98.
- FRANKEN, S.W.A. (1931)** Treatment of sensitive cervices of teeth. *Journal of the American Dental Association* 18, 156-163.
- FRENTZEN, M. & KOORT, H.T. (1990)** Lasers in dentistry. New possibilities with advancing laser technology. *International Dental Journal* 40, 23-332.
- FRY, A.E., WATKINS, R.F. & PHATAK, N.M. (1960)** Topical use of corticosteroids for the relief of pain sensitivity of dentine and pulp. *Oral Surgery, Oral Medicine, Oral Pathology* 13, 594-597.
- FRY, A.E., NEELY, A.R., RUHLMAN, D.C. & PHATAK, N.M. (1965)** Topical use of corticosteroids for the relief of pain sensitivity of dentin and pulp. Clinical observations. *Journal of Oral Therapeutics and Pharmacology* 2, 88-93.
- FUKOMOTO, C., YANAGAWA, Y., TAKASE, Y., ISHIKAWA, T., KURIYAMA, S. & MUSHA, Y. (1987)** The effectiveness of the fluoride iontophoresis. Using the pulse current. *Japanese Journal of Conservative Dentistry* 30, 1281-1288.
- FUSAYAMA, T. (1988)** Etiology and treatment of sensitive teeth. *Quintessence International* 19, 921-925.
- GANGAROSA, L.P. (1981)** Iontophoretic application of fluoride by tray techniques for desensitization of multiple teeth. *Journal of the American Dental Association* 102, 50-52.
- GANGAROSA, L.P. (1986)** Letters to the Editor. Fluoride iontophoresis for tooth desensitization. *Journal of the American Dental Association* 112, 808-810.
- GANGAROSA, L.P., BUETTNER, A.L., BAKER, W.P., BUETTNER, B.K. & THOMPSON, W.O. (1989)** Double-blind evaluation of duration of dentin sensitivity reduction by fluoride iontophoresis. *General Dentistry* July/August: 316-319.

- GANGAROSA, L.P., GADO, M. & BARATZ, R.S. (1985) Occlusion of dentinal tubules by iontophoretic application of sodium fluoride. *Journal of Dental Research* 64, 309 (Abstract no. 1212).
- GANGAROSA, L.P., HEUER, G.A., PARK, N.H., HAYES, B.B., LITTLE, C.K., BAKER, G.W. & SMITH, M.A. (1978) Desensitizing hypersensitive dentin by iontophoreseis with fluoride. *New York State Dental Journal* 44, 92-94.
- GANGAROSA, L.P. & McRAE, K. (1985) Fluoride iontophoresis as an aid in the diagnosis and treatment of reversible pulpitis. A case study. *Compendium of Continuing Education in Dentistry* 6, 696-702.
- GANGAROSA, L.P. & PARK, N.H. (1978) Practical considerations in iontophoresis of fluoride for desensitizing dentin. *Journal of Prosthetic Dentistry* 39, 173-178.
- GAO, F., WYBORN, L.E., STARCKE, E.N. & CHAN, J.T. (1991) Topical versus iontophoretic application of desensitizing agents. *Journal of Dental Research* 70, 475 (Abstract no. 1669).
- GARBEROGLIO, R. & BRÄNNSTRÖM, M. (1976) SEM investigation of human dentinal tubules. *Archives of Oral Biology* 21, 355-362.
- GARCIA-GODOY, F., GARCIA-GODOY, F., DE VIZIO, W., VOLPE, A.R., FERLAUTO, R.J. & MILLER, J.M. (1990) Effect of a triclosan/copolymer/fluoride dentifrice on plaque formation and gingivitis. A 7-month clinical study. *American Journal of Dentistry* 3, 15-26.
- GARNICK, J., HOFFMAN, L. & PASHLEY, D. (1992) Study of dentinal tubule replication. *Journal of Dental Research* 71, 124 (Abstract no. 148).
- GAZELIUS, B. & OLGART, L. (1980) Vasodilatation in the dental pulp produced by electrical stimulation of the inferior alveolar nerve in the cat. *Acta Physiologica Scandinavica* 108, 181-186.
- GEDALIA, I., SHULMAN, L.B., ALBERT, M., GOLDBERGER, P. & SCHIACKI, I. (1971) Fluoride content in root layers of fluoride-immersed teeth. *Pharmacology and Therapeutics in Dentistry*. 1, 151-156.
- GEDALIA, I., ALMOG, D. & YARIV, S. (1977) Effects of strontium and fluoride uptakes on the solubility of powdered enamel. *Caries Research* 11, 287-292.

- GEDALIA, I., BRAYER, L. KALTER, N., RICHTER, M. & STABHOLTZ, A. (1978) The effect of fluoride and strontium application on dentine, in vivo and in vitro studies. *Journal of Periodontology* 49, 269-272.
- GEDALIA, I., YARIV, S., BRAYER, L. & GREENBAUM, M. (1976) Strontium uptake by powdered and intact human root dentine. *Archives of Oral Biology* 21, 413-416.
- GESELL, A., ILG, F.L. & AMES, L.B. (1956) *Youth. The Years from Ten to Sixteen*. Hamish Hamilton, London.
- GOODIS, H.E., WHITE, J.M., YEE, K.J. & FULLER, N.D. (1992) Effect of the Nd:YAG on dentin permeability. *Journal of Dental Research* 71, 162 (Abstract no. 452).
- GOODIS, H.E., GAMM, B.M. & WATANABLE, L.G. (1989) Pulp chamber temperature changes with visible light cured composite restorations. *Journal of Dental Research* 68, 268 (Abstract no 691).
- GORMAN, W.J. (1967) Prevalence and etiology of gingival recession. *Journal of Periodontology* 38, 316-322.
- GOTTLIEB, B. (1947) *In Dental Caries. Its Etiology, Pathology, Clinical Aspects and Prophylaxis*. Henry Kimpton, London Pp 229-236.
- GRABENSTETTER, R.J., BROGE, R.W., JACKSON, F.L & RADIKE, A.W. (1958) The measurement of the abrasion of human teeth by dentifrice abrasives. A test utilizing radioactive teeth. *Journal of Dental Research* 37, 1060-1068.
- GRACELY, R.H. (1980) Pain measurement in man. In. *Pain discomfort and humanitarian care*. Ng, L.K.Y. & Bonica, J.J. (eds) New York, Elsevier North Holland Pp 111-138.
- GRACELY, R.H., McGRATH, P. & DUBNER, R. (1978) Ratio scales of sensory and affective verbal pain descriptors. *Pain* 5, 5-18.
- GRAF, W. & BJÖRLIN, G. (1951) Diameters of nerve fibres in human tooth pulps. *Journal of the American Dental Association* 43, 186-193.
- GRAF, H. & GALASSE, R. (1977) Morbidity, prevalence and intraoral distribution of hypersensitive teeth. *Journal of Dental Research* 56 (Special Issue A), 162 (Abstract no. 479).
- GRANT, D.A., STERN, I.B. & EVERETT, F.G. (1972) In: *Orban's*

- Periodontics, 4th Edition, C.V. Mosby Company, Saint Louis, Pp 414-416.
- GRANT, D.A., STERN, I.B. & EVERETT, F.G. (1979)** In. Orban's Periodontics, 5th Edition, C.V. Mosby Company, Saint Louis, Pp 527-570.
- GREEN, B.L., GREEN, M.L., & McFALL, W.T. (1977)** Calcium hydroxide and potassium nitrate as desensitizing agents for hypersensitive root surfaces. *Journal of Periodontology* 48, 667-672.
- GREENE, J.C. & VERMILLION, J.R. (1960)** The oral hygiene index: A method of classifying oral hygiene status. *Journal of the American Dental Association* 61, 172-178.
- GREENHILL, J.D. & PASHLEY, D.H. (1981)** The effects of desensitizing agents on the hydraulic conductance of human dentin *in vitro*. *Journal of Dental Research* 60, 686-698.
- GRØN, P. & CASLAVSKA, V. (1981)** Fluoride deposition in enamel from monofluorophosphate application. *Caries Research* 15, 90-97.
- GROSSMAN, L.I. (1935)** A systematic method for the treatment of hypersensitive dentine. *Journal of the American Dental Association* 22, 592-602.
- GRÜSSER, O.J., KOLLMANN, W. & MIJATOVIC, E. (1982)** Evidence for cold perception from human teeth. In: *Anatomical, Physiological and Pharmacological Aspects of Trigeminal Pain*. Matthews, B. & Hill, R.G. (eds), Amsterdam, Excerpta Medica Pp 77-95.
- GUNJI, T. (1982)** Morphological research on the sensitivity of dentin. *Archives Histology of Japan* 45, 45-67.
- GUO-HUA, L. & MORIMOTO, M. (1991)** Magnesium sulphate as a new desensitizing agent. *Journal of Oral Rehabilitation* 18, 363-372.
- GURNEY, B.F. (1970)** Chemotherapy in dental practice. Corticosteroids: Part four. *Dental Digest* 76, 283-285.
- GUTENTAG, H. (1965)** The effect of strontium chloride on peripheral nerves in comparison to the stabilizer and labilizer components. *Penn Dental Journal* 68, 37-43.
- GWINNETT, A.J. (1988)** Aluminum oxalate for dentin bonding. An SEM study. *American Journal of Dentistry* 1, 5-8.
- GYSI, A. (1900)** An attempt to explain the sensitiveness of dentine.

British Dental Journal 43, 865-868.

HAEGERSTAM, G. (1976) The origins of impulses from dental cavities in the tooth of the cat. *Acta Physiologica Scandinavica* 97, 121-128.

HAEGERSTAM, G., OLGART, L. & EDWALL, L. (1975) The excitatory action of acetylcholine on intradental sensory units. *Acta Physiologica Scandinavica* 93, 113-118.

HAHN, M.S. & KIM, J. B. (1991) A study on the methods for assessment of dentifrice abrasivity. *Journal of Dental Research* 70, 810 (Abstract no. 0-25).

HALL, E.H., TEREZHALMY, G.T. & PELLEU, G.B. (1986) A set of descriptors for the diagnosis of dental pain syndromes. *Oral Surgery, Oral Medicine, Oral Pathology* 61, 153-157.

HANSEN, S.E., SWIFT, E.J. & KRELL, K.V. (1991) Effect of dentin adhesives on permeability. *Journal of Dental Research* 70, 393 (Abstract no. 1021).

HANSSON, R.E. (1987) The assessment of the subjective nature of the pain associated with cervical root dentin hypersensitivity and the evaluation of the effectiveness of potassium oxalate in the reduction of cervical root dentin hypersensitivity. (Thesis) Ann Arbor, Michigan, University of Michigan.

HANSSON, R.E., BYE, F.L. & SMITH, B.A. (1988) Four different pain rating scales used to evaluate dentin hypersensitivity. *Journal of Dental Research* 67 (Special Issue), 292 (Abstract no. 1433).

HARRIMAN, G.B. (1872) Is the dentine tubular? *Dental Register* 26, 363-367.

HARRIS, R. & CURTIN, J.H. (1976) Dentine hypersensitivity. *Australian Dental Journal* 21, 165-169.

HARRIS, R., SCHAMSCHULA, R.G., GREGORY, G., ROOTS, M. & BEVERAGE, J. (1967) Observations on the cariostatic effects of calcium sucrose phosphate in a group of children aged 5-17 years. *Australian Dental Journal* 12, 105-113.

HARRIS, R., SCHAMSCHULA, R.G., BEVERAGE, J. & GREGORY, G. (1968) Observations on the cariostatic effects of calcium sucrose phosphate in

a group of children aged 5-17 years. Australian Dental Journal 13, 32-39.

HARRIS, R., SCHAMSCHULA, R.G., BEVERAGE, J. & GREGORY, G. (1969)

Observations on the cariostatic effects of calcium sucrose phosphate in a group of children aged 5-17 years. Australian Dental Journal 14, 42-49.

HASTREITER, R.J. (1989) Is 0.4% stannous fluoride gel an effective agent for the prevention of oral diseases? Journal of the American Dental Association 118, 205-208.

HAUGEJORDEN, C. & NORD, A. (1991) A three year trial of the fluoride containing varnishes 'Carex' and Duraphat®. Journal of Dental Research 70, 833 (Abstract no. 77).

HAUGEN, E. & JOHANSEN, J.R. (1988) Tooth hypersensitivity. A case report including SEM studies. Journal of Clinical Periodontology 15, 399-401.

HAZEN, S.P., VOLPE, A. R. & KING, W.J. (1968) Comparative desensitizing effect of dentifrices containing sodium monofluorophosphate, stannous fluoride and formalin. Periodontics 6, 230-232.

HEFFERREN, J.J. (1976) A laboratory method for assessment of dentifrice abrasivity. Journal of Dental Research 55, 563-573.

HEFT, M.W. & PARKER, S.R. (1984) An experimental basis for revising the category rating scale for pain. Pain 19, 153-161.

HELD, A.J. & BAUD, C.A. (1955) Innervation of the dental organ. Studied by new techniques. Oral Surgery, Oral Medicine, Oral Pathology 8, 1262-1269.

HEMBREE, M.E. & HEMBREE, J.H. (1977) Relative abrasiveness of dentifrices. Dental Hygiene 51, 253-255.

HERNANDEZ, F., MOHAMMED, C., SHANNON, I., VOLPE, A., KING, W. (1972) Clinical study evaluating the desensitizing effect and duration of two commercially available dentifrices. Journal of Periodontology 43, 367-372.

HEYMANN, H.O., STURDEVANT, J.R., BRUNSON, W.D., WILDER, A.D., BAYNE, S.C. & SLUDER, T.B. (1987) Six month clinical study of dentinal

adhesives in class v eroded lesions. Journal of Dental Research 66, 268 (Abstract no. 1294).

HIATT, W.E. & JOHANSEN, E. (1972) Root preparation I. Obturation of dentinal tubules in treatment of root hypersensitivity. Journal of Periodontology 43, 373-380.

HIRVONEN, T. & NÄRHI, M.V.O. (1986) The effect of dentinal stimulation on pulp nerve function and pulp morphology in the dog. Journal of Dental Research 65, 1290-1293.

HIRVONEN, T.J., NÄRHI, M.V.O. & HAKUMAKI, M.O.K. (1984) The excitability of dog pulp nerves in relation to the condition of dentin surface. Journal of Endodontics 10, 294-298.

HODOSH, M. (1974) A superior desensitizer - potassium nitrate. Journal of the American Dental Association 88, 831-832.

HOLLAND, G.R. (1975) The dentinal tubule and odontoblast process in the cat. Journal of Anatomy 120, 169-177.

HOLLAND, G.R. (1980) Microtubule and microfilament populations of cell processes in the dental pulp. Anatomical Record 198, 421-426.

HOLLAND, G.R. (1985) The odontoblast process form and function. Journal of Dental Research 64 (Special Issue) 499-514.

HOLLAND, G.R. (1990) Role of the odontoblast process. In: Dynamic aspects of the Pulp, Molecular Biology, Pharmacology and Pathophysiology. Inoki, R., Kudo, T. & Olgart, L.M. (eds) Chapman & Hall, London Pp 73-95.

HORIUCHI, H. (1991) Dentinal sensation. Its mechanism and clinical significance. Journal of Dental Research 70, 806 (Abstract no. S-2).

HORIUCHI, H. & MATTHEWS, B. (1971) Action potentials from a model tooth. Archives of Oral Biology 20, 17-18.

HORIUCHI, H. & MATTHEWS, B. (1973) In vitro observations on fluid flow through human dentine caused by pain producing stimuli. Archives of Oral Biology 18, 275-294.

HORIUCHI, H. & MATTHEWS, B. (1974) Evidence on the origin of impulses recorded from dentine in the cat. Journal of Physiology 243, 797-829.

HORIUCHI, H. & MATTHEWS, B. (1975) Action potentials from a model

tooth. Archives of Oral Biology 20, 17-18.

HORIUCHI, H. & MATTHEWS, B. (1976) Responses of intradental nerves to chemical and osmotic stimulation of dentine in the cat. Pain 2, 49-59.

HOVGAARD, O., LARSEN, M.J. & FEJERSKOV, O. (1988) Hypersensitive root surfaces - Effect of oral hygiene and topical application of NaF and Ca(OH)₂. Journal of Dental Research 67, 329 (Abstract no. 1734).

HOYT, W.H. & BIBBY, B.G. (1943) Use of sodium fluoride for desensitizing dentin. Journal of the American Dental Association 30, 1372-1376.

HUME, W.R. (1984a) Effect of eugenol respiration and division of human pulp, mouse fibroblasts and liver cells in vitro. Journal of Dental Research 63, 1262-1265.

HUME, W.R. (1984b) An analysis of the release and the diffusion through dentin of eugenol from zinc oxide-eugenol mixtures. Journal of Dental Research 63, 881-884.

HUSKISSON, E.C. (1974) Measurement of pain. Lancet 2, 1127-1131.

IANZANO, J.A. & GWINNETT, A.J. (1992) Polymeric sealing of dentinal tubules to control sensitivity. Journal of Dental Research 71, 205 (Abstract no. 798).

ISHIKAWA, S. (1960) Über das lymphsystem des zahnes (On the lymphatic system of teeth). Zeitschrift für Zellforschung und mikroskopische Anatomie 52, 140-149.

ISHIKAWA, S. (1969) A clinico-histological study on the hypersensitivity of dentin. Journal of the Japanese Stomatology Society 36, 68-88, 278-298. (In Japanese).

ITO, S., TONZETICH, J., CLARK, D.C. & HATA, Y (1991) Evaluation of remineralizing solution on sensitive teeth by replica technique. Journal of Dental Research 70, 756 (Abstract no. 129).

IZAWA, T., LIU, M., DÖRSCHER-KIM, J., KIM, S. & KISHI, Y. (1991) Structural and functional changes in dentin/pulp complex in response to Nd:YAG lasing. Proceedings of International Conference on Pathobiology of the Dentin/Pulp Complex. University of North Carolina 25-29 May, Pp 9-10.

- JACKSON, R.J., DUKE, S.A. & WICKS, M.A. (1989) The evaluation of two dentifrices formulated for the treatment of dentine hypersensitivity. *Journal of Dental Research* 68, 602 (Abstract no. 346).
- JACKSON, R.J., DUKE, S.A., WICKS, M.A. & McDONALD, F.E. (1990) The effects of antisensitivity dentifrices on subjects consuming acid diets. *Journal of Dental Research* 69, 988 (Abstract no. 268).
- JAVID, B., BARKHORDAR, R.A. & BHINDA, S.V. (1987) Cyanoacrylate - A new treatment for hypersensitive dentin and cementum. *Journal of the American Dental Association* 114, 486-488.
- JENSEN, A.L. (1964) Hypersensitivity controlled by iontophoresis, double blind clinical investigation. *Journal of the American Dental Association* 68, 216-225.
- JENSEN, M.E. & DOERING, J.V. (1987) A comparative study of two clinical techniques for treatment of root surface hypersensitivity. *General Dentistry* 35, 128-132.
- JENSEN, M.E. & KOHOUT, F. (1988) The effect of a fluoridated dentifrice on root and coronal caries in an older adult population. *Journal of the American Dental Association* 117, 829-832.
- JESKE, A.H. (1984) Reduction of dentin hypersensitivity using fluoride iontophoresis. *Illinois Dental Journal* 53, 179-180.
- JOHANSEN, E. & PARKS, H.F. (1962) Electron microscopic observation on sound human dentine. *Archives of Oral Biology* 7, 185-193.
- JOHNSEN, D. & JOHNS, S. (1978) Quantification of nerve fibres in the primary and permanent canine and incisor teeth in man. *Archives of Oral Biology* 23, 825-829.
- JOHNSEN, D.C. (1985) Innervation of teeth qualitative, quantitative and development assessment. *Journal of Dental Research* 64 (Special Issue) 555-563.
- JOHNSEN, D.C. (1990) Innervation of teeth: developmental aspects. In. *Dynamic Aspects of Dental Pulp, Molecular Biology, Pharmacology and Pathophysiology*. Inoki, R., Kudo, T. & Olgart, L.M. (eds) Chapman & Hall, London Pp 3-28.
- JOHNSON, G. & BRÄNNSTRÖM, M. (1974) The sensitivity of dentine -

Changes in relation to conditions at exposed tubule apertures. *Acta Odontologica Scandinavica* 32, 29-38.

JOHNSON, G., OLGART, L. & BRÄNNSTRÖM, M. (1973) Outward fluid flow in dentine under a physiologic pressure gradient experiment *in vitro*. *Oral Surgery, Oral Medicine, Oral Pathology* 35, 238-248.

JOHNSON, R.H., ZULQAR-NAIN, B.J. & KOVAL, J.J. (1982) The effectiveness of an electro-ionizing toothbrush in the control of dentinal hypersensitivity. *Journal of Periodontology* 53, 353-359.

JOHNSTON, A.D., ASMUSSEN, E. & BOWEN, R.L. (1989) Substitutes for N-phenylglycine in adhesive bonding to dentin. *Journal of Dental Research* 68, 1337-1344.

JORKJEND, I. & TRONSTAD, L. (1972) Treatment of hypersensitive teeth by calcium hydroxide. *Scandinavian Journal of Dental Research* 80, 264-266.

JOYCE, C.R.B., ZUTSHI, D.W., HRUBES, V. & MASON, R.M. (1975) Comparison of fixed interval and visual analogue scales for rating chronic pain. *European Journal of Clinical Pharmacology* 8, 415-420.

JYVÄSJÄRVI, E. (1986) Electrophysical studies of afferent C-fibre innervation in the dental pulp. Thesis University of Helsinki, Finland.

JYVÄSJÄRVI, E. & KNIFFKI, K.D. (1987) Cold Stimulation of Teeth: A Comparison between the responses of cat intra-dental A δ and C fibres and human sensation. *Journal of Physiology* 391, 193-207.

JYVÄSJÄRVI, E. & KNIFFKI, K.D. (1992) Studies on the presence and functional properties of afferent C-fibres in the cat's dental pulp. *Proceedings of the Finnish Dental Society* 88 (Supplement 1), 533-542.

KAMINSKI, K., TUNG, M. & TAKAGI, K. (1990) Effects of oxalate and calcium phosphate solutions on dentin tubule obstruction. *Journal of Dental Research* 69, 168 (Abstract no. 480).

KANAPKA, J.A. (1982) A new agent. *Compendium of Continuing Education in Dentistry Supplement* 3, 118-120.

KANAPKA, J.A. (1990) Over-the-Counter dentifrices in the treatment of tooth hypersensitivity. Review of clinical studies. *Dental Clinics of North America* 34, 545-560.

KANAPKA, J.A. & COLUCCI, S.V. (1986) Clinical evaluation of dentinal

hypersensitivity. A comparison of methods. Endodontics & Dental Traumatology 2, 157-164.

KANOUSE, M.C. & ASH, M.M. (1969) The effectiveness of a sodium monofluorophosphate dentifrice on dental hypersensitivity. Journal of Periodontology 40, 38-40.

KARLSSON, U.L. & PENNEY, D.A. (1975) Natural desensitization of exposed tooth roots in dogs. Journal of Dental Research 54, 982-986.

KAUFMAN, C., PELZNER, R.B., SOELBERG, K.B. & BOGDAN, M.S. (1982) An evaluation of the protective properties of a new varnish. Quintessence International 6, 691-695.

KEELE, K.D. (1948) The pain chart. Lancet, Volume II, 6-8.

KERN, D.A., McQUADE, M.J., SCHEIDT, M.J., HANSON, B. & VAN DYKE, T.E. (1989) Effectiveness of sodium fluoride on tooth hypersensitivity with or without iontophoresis. Journal of Periodontology 60, 386-389.

KERNS, D.G., SCHEIDT, M.J., PASHLEY, D.H., HORNER, J.A., STRONG, S.L. & VAN DYKE, T.E. (1991) Dentinal tubule occlusion and root hypersensitivity. Journal of Periodontology 62, 421-428.

KIM, S. (1986a) Thermal stimuli in dentinal sensitivity. Endodontics & Dental Traumatology 2, 138-140.

KIM, S. (1986b) Hypersensitive teeth. Desensitization of pulpal sensory nerves. Journal of Endodontics 12, 482-485.

KIM, S. (1990) Neurovascular interactions in the dental pulp in health and inflammation. Journal of endodontics 16, 48-53.

KIM, S., LIU, M-T. & DÖRSCHER-KIM, J. (1986) Comparison of intra arterial bolus injection and slow infusion of substance P on pulpal blood flow in dogs. Journal of Dental Research 66, 186 (Abstract no. 150).

KIM, S., TROWBRIDGE, H. & DÖRSCHER-KIM, J. (1985) The effects of substance P on pulpal hemodynamics in dogs. Journal of Dental Research 65, 310 (Abstract no. 1216).

KIM, S., DÖRSCHER-KIM, J.E., LIU, M. & GRAYSON, A. (1992) Functional alterations in pulpal microcirculation in response to various dental procedures and materials. Proceedings of the Finnish Dental Society

88 (Supplement 1), 65-71.

KIMMELMAN, B.B., KING, R.J., LORENZO, J., SOLOMON, M., ZAFRAN, J.N. & ZAYON, G.M. (1969) Clinical testing of a desensitizing dentifrice - Design and findings. *Journal of the New Jersey State Dental Society* 40, 279-288.

KLAUS, B. & GANGAROSA, L.P. (1986) The long-term effect of sodium fluoride iontophoresis for treatment of hypersensitive dentin. *Quintessenz* 38, 81-87.

KLEINBERG, I. (1986) Dentinal hypersensitivity, Part II: Treatment of sensitive dentin. *Compendium of Continuing Education in Dentistry* 7, 280-284.

KLEINBERG, I., KAUFMAN, H.W. & CONFESSORE, F. (1990) Methods of measuring tooth hypersensitivity. *Dental Clinics of North America* 34, 515-529.

KOLLMANN, W. & MATTHEWS, B. (1982) Responses of intradental nerves to thermal stimulation in the cat. In *Anatomical, Physiological & Pharmacological Aspects of Trigeminal Pain*. Matthews, B. & Hill, R.G. (eds) *Excerpta Medica*, Amsterdam Pp 51-65.

KOWITZ, G. & MENG, Z. (1989) Tartar control toothpaste and dental hypersensitivity. *Journal of Dental Research* 68, 313 (Abstract no. 1053).

KOZAM, G. (1977) The effects of eugenol on nerve transmission. *Oral Surgery, Oral Medicine, Oral Pathology* 44, 799-805.

KRAMER, I.R.H. (1955) The relationship between dentine sensitivity and movement in the contents of the dentinal tubules. *British Dental Journal* 98, 391-392.

KRAUSER, J.T. (1986) Hypersensitive teeth. Part II. Treatment. *Journal of Prosthetic Dentistry* 56, 307-311.

KROEGER, D.C. (1968) Possible Role of Neurohumoral Substances in the Pulp Biology of the Dental Pulp Organ. A symposium, Finn, S.B. (ed) *Birmingham, University of Alabama Press* Pp 333-346.

KROEGER, D.C., GONZALES, F. & KRIVOVY, W. (1961) Transmembrane potentials of cultured mouse dentinal pulp cells. *Proceedings of the*

Society of Experimental Medicine 108, 134-136.

KUN, L. (1976) Etude biophysique des modifications des tissus dentaires provoquées par l'application locale de strontium. Schweizerische Monatsschrift für Zahnheilkunde 86, 661-676.

KUKLETOVÁ, M., ZAHŘÁDKA, J. & LUCÁŠ, Z. (1968) Monoaminergic and cholinergic nerve fibres in the human dental pulp. Histochemie 16, 154-158.

LAFLECHÉ, R.G., FRANK, R.M. & STEUER, P. (1985) The extent of the human odontoblast process as determined by transmission electron microscopy: the hypothesis of a retractable suspensor system. Journal de Biologie Buccale 13, 293-305.

LAMB, J.F., INGRAM, C.G., JOHNSTON, I.A. & PITMAN, R.M. (1980) In. Essentials of Physiology, Blackwell Scientific Publications, Oxford Pp 31-32.

LANGLELAND, K., DOWDEN, W.E., TRONSTAD, L. & LANGLELAND, L. (1971) Human pulp changes of iatrogenic origin. Oral Surgery, Oral Medicine, Oral Pathology 32, 943-980.

LAUFER, B. MAYER, I., GEDALIA, I., DEUTSCH, D., KAUFMAN, H.W. & TAL, M. (1981) Fluoride-uptake and fluoride residual of fluoride-treated human root dentine in vitro determined by chemical, scanning electron microscopy and X-ray diffraction analyses. Archives of Oral Biology 26, 159-163.

LAWSON, B.F. & HUFF, T.W. (1966) Desensitization of teeth with a topically applied glucocorticoid drug. A preliminary study. Journal of Oral Therapeutics & Pharmacology 2, 295-299.

LECOINTRE, C., APIOU, J., MARTY, P. & POITOU, P. (1986) Controlled trial of the action of a toothpaste containing nicomethanol hydrofluoride in the treatment of dentine hypersensitivity. Journal of International Medicine Research 14, 217-222.

LEE, I.K., HAN, S.B., KO, J.S. & KIM, H.M. (1991) Clinical & scanning electron microscopic observations after treatment of dentine hypersensitivity in vivo. Journal of Dental Research 70, 816 (Abstract no. 0-79).

- LEFKOWITZ, W. (1962) Pulp response to ionization. *Journal of Prosthetic Dentistry* 12, 966-976.
- LEFKOWITZ, W., BURDICK, B.A. & MOORE, D.L. (1963) Desensitization of dentin by bioelectric induction of secondary dentin. *Journal of Prosthetic Dentistry* 13, 940-949.
- LEVIN, M.P., YEARWOOD, L.L., CARPENTER, W.N. (1973) The desensitizing effect of calcium hydroxide and magnesium hydroxide on hypersensitive dentin. *Oral Surgery, Oral Medicine, Oral Pathology* 35, 741-746.
- LIBERMAN, R., BEN-AMAR, A., SEREBRO, L. & AVISHAI, S. (1986) Marginal seal of composite resin restorations - A comparative study using two new sealing materials (an *in vitro* study). *Dental Medicine* 4, 17-20.
- LILIENTHAL, B., NAPPER, D.H. & SMYTHE, B.M. (1968) The hardening and softening of human tooth enamel. *Australian Dental Journal* 13, 219-230.
- LILJA, J. (1979) Innervation of different parts of the predentine and dentine in young human premolars. *Acta Odontologica Scandinavica* 37, 339-346.
- LILJA, J. (1980) Sensory differences between crown and root dentine in human teeth. *Acta Odontologica Scandinavica* 38, 285-294.
- LILJA, J., NORDENVALL, K.J. & BRÄNNSTRÖM, M. (1982) Dentine sensitivity, odontoblasts and nerves under dessicated or infected experimental cavities. *Swedish Dental Journal* 6, 93-103.
- LINDEN, L.A. (1968) Microscopic observations of fluid flow through cementum and dentine. *Odontologisk Revy* 19, 367-381.
- LIU, M., PERTL, C., Markowitz, K., DÖRSCHER-KIM, J. & KIM, S. (1992) The effects of capsaicin on pulpal blood flow. *Proceedings of the Finnish Dental Society* 88 (Supplement 1), 463-467.
- LÖE, H. (1967) The Gingival Index, the Plaque Index and the Retention Index Systems. *Journal of Periodontology* 38, 610-616.
- LÖE, H. & SILNESS, J. (1963) Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odontologica Scandinavica* 21, 533-551.
- LÖE, H., ANERUD, A., BOYSEN, H. & SMITH, M. (1978) The natural history of periodontal disease in man. The rate of periodontal destruction before 40 years of age. *Journal of Periodontology* 49, 607-620.

- LOW, T. (1981)** The treatment of hypersensitive cervical abrasion cavities using ASPA cement. *Journal of Oral Rehabilitation* 8, 81-89.
- LUKOMSKY, E.H. (1941)** Fluorine therapy for exposed dentine and alveolar atrophy. *Journal of Dental Research* 20, 649-659.
- LUNDY, T. & STANLEY, H.R. (1969)** Correlation of pulpal histopathology and clinical symptoms in human teeth subjected to experimental irritation. *Oral Surgery, Oral Medicine, Oral Pathology* 27, 187-201.
- LUSSI, A., SCHICK, A. & HOTZ, P. (1989)** Effect of different fluoride treatments on dental erosion. *Journal of Dental Research* 68, 634 (Abstract no. 200).
- LUTINS, N.D., GRECO, G.W. & MCFALL, W.T. (1984)** Effectiveness of sodium fluoride on tooth hypersensitivity with and without iontophoresis. *Journal of Periodontology* 55, 285-288.
- MacGREGOR, I.D.M. & RUGG GUNN, A.J. (1979)** A survey of toothbrushing sequence in children and young adults. *Journal of Periodontal Research* 14, 225-230.
- MANOCHEHR-P, M., BHAT, M. & BISSADA, N. (1984)** Clinical evaluation of two potassium nitrate toothpastes for the treatment of dental hypersensitivity. *Periodontal Case Reports* 6, 25-30.
- MARKOWITZ, K., BILOTTO, G. & KIM, S. (1991)** Decreasing intradental nerve activity in the cat with potassium and divalent cations. *Archives of Oral Biology* 36, 1-7.
- MARKOWITZ, K. & KIM, S. (1985)** The effects of various ionic solutions on pulpal nerve sensitivity. *Journal of Dental Research* 64, 309 (Abstract no. 1213).
- MARKOWITZ, K. & KIM, S. (1990)** Hypersensitive teeth. Experimental studies of dentinal desensitizing agents. *Dental Clinics of North America* 34, 491-501.
- MARKOWITZ, K. & KIM, S. (1992)** The role of selected cations in the desensitization of intradental nerves. *Proceedings of the Finnish Dental Journal* 88 (Supplement 1), 39-54.
- MARKOWITZ, K., NÄRHI, M., NGASSAPA, D. & KIM, S. (1989)** The effect of KCl on intradental nerve activity evoked by hypertonic NaCl solution

and natural stimuli. Journal of Dental Research 68, 295 (Abstract no. 1492).

MASON, S., LEVAN, A., CRAWFORD, R., FISHER, S. & GAFFAR, A. (1989)

Effect of tartar control dentifrices on dentine tubules in vitro.

Journal of Dental Research 68, 313 (Abstract no. 1054).

MASSLER, M. (1955) Desensitization of cervical cementum and dentine by sodium silicofluoride. Journal of Dental Research 34, 761-762.

MATSUMOTO, K., IZUMI, M. & NAGASAWA, H. (1980) Scanning electron microscopic study on the hypersensitivity of dentin. Japanese Journal of Conservative Dentistry 23, 247-251.

MATSUMOTO, K., OYAMATA, T., SHIRASUKA, T. & WAKABAYASHI, H. (1990)

Study on the replica of dentin tubules by silicon-resin method. Journal of Showa University of Dentistry 10, 203-205.

MATTHEWS, B. (1968) Cold sensitive and heat-sensitive nerves in teeth. Journal of Dental Research 47, 974.

MATTHEWS, B. (1970) Nerve impulses recorded from dentine in the cat. Archives of Oral Biology 15, 523-530.

MATTHEWS, B. (1972) Sensory mechanisms in dentine. Proceedings of the Royal Society of Medicine 65, 493-495.

MATTHEWS, B. (1977) Responses of intradental nerves to electrical and thermal stimulation of teeth in dogs. Journal of Physiology 264, 641-664.

MATTHEWS, B. (1992) Sensory physiology: a reaction. Proceedings of the Finnish Dental Society 88 (Supplement 1), 529-532.

MATTHEWS, B. & HOLLAND, G.R. (1975) Coupling between nerves in teeth. Brain Research 98, 354-358.

MATTHEWS, B. & SEARLE, B.N. (1976) Electrical stimulation of teeth. Pain 2, 245-251.

MAYER, R. (1964) Beitrag zur Behandlung über empfindlicher Zähne. Das Deutsche Zahnärzteblatt 19, 172-175.

MAZETTI, D. & TOLEDO, O. (1971) Secondary mineralization of rat healthy dentin by means of calcium hydroxide. A fluorescence study. Journal of Child Dentistry 38, 247-251.

- MAZOR, Z., BRAYER, L., FRIEDMAN, M. & STEINBERG, D. (1989) Treatment of dentinal hypersensitivity by SRD containing SrCl_2 topically applied. *Journal of Dental Research* 68, 656 (Abstract no. 6).
- McBRIDE, M.A., GILPATRICK, R.O. & FOWLER, W.L. (1991) The effectiveness of sodium fluoride iontophoresis in patients with sensitive teeth. *Quintessence International* 22, 637-640.
- McFALL, W.T. (1986) A review of the active agents available for treatment of dentinal hypersensitivity. *Endodontics & Dental Traumatology* 2, 141-149.
- McFALL, W.T. & HAMRICK, S.W. (1987) Clinical effectiveness of a dentifrice containing fluoride and a citrate buffer system for the treatment of dentinal sensitivity. *Journal of Periodontology* 58, 701-705.
- McFALL, W.T. & MORGAN, W.C. (1985) Effectiveness of a dentifrice containing formalin and sodium monofluoro-phosphate on dental hypersensitivity. *Journal of Periodontology* 56, 288-292.
- McGRATH, P.A. (1986) The measurement of human pain. *Endodontics & Dental Traumatology* 2, 124-129.
- MEFFERT, R.M. & HOSKINS, S.W. (1964) Effect of a strontium chloride dentifrice in relieving dental hypersensitivity. *Journal of Periodontology* 35, 232-235.
- MELLBERG, J.R. (1991) Fluoride dentifrices. Current status and prospects. *International Dental Journal* 41, 9-16.
- MELZACK, R. (1973) *The puzzle of pain*. Basic Books, New York.
- MELZACK, R. (1975) The McGill Pain Questionnaire: major properties and scoring methods. *Pain* 1, 277-299.
- MELZACK, R. & WALL, P. (1988) *The challenge of pain* Penguin books, Pp 34-46.
- MIDDA, M. (1990) NdYAG lasers. Their use in dentistry. *Dental Practice* 28, 1-3.
- MIDDA, M. & RENTON-HARPER, P. (1991) *Lasers in Dentistry*. British Dental Journal 170, 343-346.
- MILLER, J.T., SHANNON, I.L., KILGORE, W.G. & BOOKMAN, J.E. (1969) Use

- of a water-free stannous fluoride-containing gel in the control of dental hypersensitivity. *Journal of Periodontology* 40, 490-491.
- MINKOFF, S. & AXELROD, S. (1987)** Efficacy of strontium chloride in dental hypersensitivity. *Journal of Periodontology* 58, 470-474.
- MINKOV, B., MARMARI, I., GEDALIA, I. & GARFUNKEL, A. (1975)** The effectiveness of sodium fluoride treatment with or without iontophoresis on the reduction of hypersensitive dentin. *Journal of Periodontology* 46, 246-249.
- MJÖR, I.A. (1967)** Histologic studies of human coronal dentine following the insertion of various materials in experimentally prepared cavities. *Archives of Oral Biology* 12, 441-452.
- MJÖR, I.A., FINN, S.B. & QUIGLEY, M.B. (1961)** The effects of calcium hydroxide and amalgam on non-carious vital dentine. *Archives of Oral Biology* 3, 283-291.
- MJÖR, I.A. & FURSETH, R. (1968)** The inorganic phase of calcium hydroxide - and corticosteroid-covered dentine. Studied by electron microscopy. *Archives of Oral Biology* 13, 755-763.
- MJÖR, I.A. & PINDBORG, J.J. (1973)** *Histology of the Human Tooth*. Copenhagen, Langkjaers Boytrykkeri.
- MOSTAFA, P. (1985)** Clinical and laboratory studies of desensitizing dentifrices. PhD Thesis, University of Wales.
- MOSTAFA, P., ADDY, M. & MORGAN, T. (1983)** Scanning electronmicroscopic, X-ray diffraction analysis, atomic absorption and fluoride probe measurements of the uptake of toothpaste ingredients onto dentine. *Journal of Dental Research* 62, 433 (Abstract no. 165).
- MOSTELLER, J.H. (1962)** Use of prednisolone in the elimination of post-operative thermal sensitivity - A clinical study. *Journal of Prosthetic Dentistry* 12, 1176-1179.
- MOSTELLER, J.H. (1963)** The ability of a prednisolone solution to eliminate pulpal inflammation. *Journal of Prosthetic Dentistry* 13, 754-760.
- MUMFORD, J.M. (1965)** Pain perception threshold and adaptation of normal human teeth. *Archives of Oral Biology* 10, 957-968.

- MUMFORD, J.M. (1967) Resistivity of human enamel and dentine. Archives of Oral Biology 12, 925-927.
- MUMFORD, J.M. (1976) Toothache and Oralfacial Pain. 2nd edition, Churchill Livingstone.
- MUMFORD, J.M. (1982) Oralfacial Pain. Aetiology, Diagnosis and Treatment. 3rd editon, Churchill Livingstone, Edinburgh.
- MUMFORD, J.M. & BJÖRN, H. (1962) Problems in electrical pulp-testing and dental algesimetry. International Dental Journal 12, 161-179.
- MUMMERY, J.H. (1924) The Microscopic and General Anatomy of the Teeth. 2nd edition. London, Humphrey Milford, Pp 209-235.
- MURTHY, K.S., TALIM, S.T. & SINGH, I. (1973) A comparative evaluation of topical application and iontophoresis of sodium fluoride for desensitization of hypersensitive dentin. Oral Surgery, Oral Medicine, Oral Pathology 36, 448-458.
- MUZZIN, K.B. & JOHNSON, R. (1989) Effects of potassium oxalate on dentin hypersensitivity *in vivo*. Journal of Periodontology 60, 151-158.
- NAPPER, D.H. & SMYTHE, B.M. (1966) The dissolution kinetics of hydroxyapatite in the presence of kink poisons. Journal of Dental Research 45, 1775-1783.
- NÄRHI, M.V.O. (1985a) The characteristics of intradental sensory units and their response to stimulation. Journal of Dental Research 64, 654-671.
- NÄRHI, M.V.O. (1985b) Dentin sensitivity. A review. Journal de Biologie Buccale 13, 75-96.
- NÄRHI, M. (1990a) The neurophysiology of the teeth. Dental Clinics of North America 34, 439-448.
- NÄRHI, M.V.O. (1990b) Intradental Sensory Units. In: Dynamic Aspects of Dental Pulp. Molecular Biology, Pharmacology and Pathophysiology. Inoki, R., Kudo, T. & Olgart, L.M. (eds) Chapman & Hall, London Pp 137-149.
- NÄRHI, M. & HAEGERSTAM (1983) Intradental nerve activity induced by reduced pressure applied to exposed dentine in the cat. Acta Physiologica Scandinavica 119, 381-386.

- NÄRHI, M. & HIRVONEN, T. (1983) Functional changes in cat nerve activity after thermal and mechanical injury of the pulp. *Proceedings of the Finnish Dental Society* 79, 162-167.
- NÄRHI, M.V.O. & HIRVONEN, T. (1987) The response of dog intradental nerves to hypertonic solutions of CaCl_2 and NaCl and other stimuli applied to exposed dentine. *Archives of Oral Biology* 32, 781-786.
- NÄRHI, M.V.O., HIRVONEN, T.J. & HAKUMÄKI, M.O.K. (1982a) Activation of intradental nerves in the dog to some stimuli applied to dentine. *Archives of Oral Biology* 27, 1053-1058.
- NÄRHI, M., HIRVONEN, T. & HAKUMÄKI, M.O. (1984) The excitability of dog pulp nerves in relation to the condition of the dentine surface. *Journal of Endodontics* 10, 294-298.
- NÄRHI, M., JYVÄSJÄRVI, E., HIRVONEN, T. & HUOPANIEMI, T. (1982b) Activation of heat-sensitive nerve fibres in the dental pulp of the cat. *Pain* 14, 317-326.
- NÄRHI, M., JYVÄSJÄRVI, E., VIRTANEN, A., HUOPANIEMI, T., NGASSAPA, D. & HIRVONEN, T. (1992a) Role of intradental A-and C- type nerve fibres in dental pain mechanisms. *Proceedings of the Finnish Dental Society* 88 (Supplement 1), 507-516.
- NÄRHI, M., KONTTURI-NÄRHI, V., HIRVONEN, T. & NGASSAPA, D. (1992b) Neurophysiological mechanisms of dentin hypersensitivity. *Proceedings of the Finnish Dental Society* 88 (Supplement 1), 15-22.
- NÄRHI, M., NGASSAPA, D., HIRVONEN, T. & KARHUNEN, V. (1988) The thresholds of dog intradental nerves to osmotic stimulation of dentine. *Journal of Dental Research* 67, 134, (Abstract no. 172).
- NÄRHI, M., KONTTURI-NÄRHI, V. & MARKKANEN, H. (1991) Sensitivity of teeth with exposed dentine to air blasts, cold and electrical stimulation. *Journal of Dental Research* 70, 448 (Abstract no. 1457).
- NAYLOR, M.N. (1961) A thermo-electric tooth stimulator. *British Dental Journal* 110, 228-230.
- NAYLOR, M.N. (1963) Studies on the mechanism of sensation to cold stimulation of human dentine. In. *Sensory Mechanisms of Dentine*. Anderson, D.J. (ed), Oxford, Pergamon press Pp 80-87.

- NAYLOR, M.N. (1968) The effect of silver nitrate and the uncooled high speed bur on dentine sensitivity. In. Dentine and pulp. Their Structures and Reactions. Symons, N.B.B. (ed), E. & S. Livingstone Ltd., Edinburgh & London Pp 247-253.
- NEIL (1850) Original reference by Blandy A.A. (1850/51) On sensitivity of teeth. American Journal of Dental Science 1, 22-28. Cited by Rosenthal, M.W. (1990)
- NORDENVALL, K.J. & BRÄNNSTRÖM, M. (1980) In vivo resin impregnation of dentinal tubules. Journal of Prosthetic Dentistry 44, 630-637.
- NORDENVALL, K.J., MALMGREN, B. & BRÄNNSTRÖM, M. (1984) Desensitization of dentin by resin impregnation: a clinical and light-microscopic investigation. Journal of Dentistry for Children 51, 274-276.
- OHNSHAUS, E.E. & ADLER, R. (1975) Methodological problems in the measurement of pain. A comparison between the verbal rating scale and the visual analogue scale. Pain 1, 379-384.
- O'LEARY, T.J., DRAKE, R.B., CRUMP, P.P. & ALLEN, M.F. (1971) The incidence of recession in young males. A further study. Journal of Periodontology 42, 264-267.
- O'LEARY, T.J., DRAKE, R.B., JIVIDEN, G.J. & ALLEN, M.F. (1968) The incidence of recession in young males. Relationship to gingival and plaque scores. Periodontics 6, 109-111.
- OLGART, L.M. (1979) Local mechanisms in dental pain. In. Mechanisms of Pain and Analgesic Compounds. Beers, R.F., & Bassett, E.G. (eds) New York, Raven Press Pp 285-294.
- OLGART, L.M. (1990) Functions of peptidergic nerves. In Dynamic Aspects of Dental Pulp, Molecular Biology, Pharmacology and Pathophysiology. Inoki, R., Kudo, T. & Olgart, L.M. (eds) Chapman & Hall, London Pp 349-362.
- OLGART, L.M., HOFELT, J., NILSSON, G. & PERNOW, B. (1977) Localisation of substance P like immunoreactivity in nerves in the tooth pulp. Pain 4, 153-159.
- ONG, G. (1983) Clinical trial of a desensitising dentifrice. MSc Thesis University of London Pp 42-49.

- ONG, G. (1986) Desensitizing Agents. A review. *Clinical Preventive Dentistry* 8, 14-18.
- ONG, G. & STRAHAN, J.D. (1989) Effect of a desensitizing dentifrice on dentinal hypersensitivity. *Endodontics and Dental Traumatology* 5, 213-218.
- ORCHARDSON, R. (1978) The generation of nerve impulses in mammalian axons by changing the concentration of the normal constituents of extracellular fluid. *Journal of Physiology* 275, 177-189.
- ORCHARDSON, R. (1985) Is calcium more effective than strontium as a desensitizing agent for dentine? In: *Current Topics in Oral Biology*, Lisney, S.J.W. & Matthews, B. (eds), Bristol, University of Bristol press Pp 205-215.
- ORCHARDSON, R. & COLLINS, W.J.N. (1984) Clinical features of 'hyper-sensitive' (HS) dentine. *Journal of Dental Research* 63, 521 (Abstract no. 282).
- ORCHARDSON, R. & COLLINS, W.J.N. (1987a) Clinical features of hypersensitive teeth. *British Dental Journal* 162, 253-256.
- ORCHARDSON, R. & COLLINS, W.J.N. (1987b) Thresholds of hypersensitive teeth to 2 forms of controlled stimulation. *Journal of Clinical Periodontology* 14, 68-73.
- ORCHARDSON, R. & LUCAS, M.L. (1991) Numerical simulation of potassium ion diffusion in dentinal tubules. *Journal of Dental Research* 70, 717 (Abstract no. 376).
- OVERMAN, P.R. (1983) Calcium hypophosphate as a root desensitizing agent. *Dental Hygiene* 57, 30, 32, 34-35.
- OYAMA, T. & MATSUMOTO, K. (1991) A clinical and morphological study of cervical hypersensitivity. *Journal of Endodontics* 17, 500-502.
- PAGHIDIWALA, A.F. (1991) Does the laser work on hard dental tissues. *Journal of the American Dental Association* 122, 79-80.
- PANOPOULOS, P. (1983) Intradental sensory nerve responses to some factors affecting dentin and pulp. Thesis Karolinska Institute, Stockholm.
- PANOPOULOS, P., GAZELIUS, B. & OLGART, L. (1983) Response of feline

intradental sensory nerves to hyperosmotic stimulation of dentin. *Acta Odontologica Scandinavica* 41, 369-375.

PASHLEY, D.H. (1984) Smear layer: physiological considerations. *Operative Dentistry Supplement* 3, 13-29.

PASHLEY, D.H. (1985a) Strategies for clinical evaluation of drugs and/or devices for the alleviation of hypersensitive dentin. In, Rowe, N.H. (ed). *Proceedings of Symposium on Hypersensitive Dentin. Origin and Management*, University of Michigan, Pp 65-88.

PASHLEY, D.H. (1985b) Dentin-predentin complex and its permeability: physiologic overview. *Journal of Dental Research* 64 (Special Issue), 613-620.

PASHLEY, D.H. (1986a) Dentin permeability, dentin sensitivity, and treatment through tubule occlusion. *Journal of Endodontics* 12, 465-474.

PASHLEY, D.H. (1986b) Sensitivity of dentin to chemical stimuli. *Endodontics & Dental Traumatology* 2, 130-137.

PASHLEY, D.H. (1990) Mechanisms of dentin sensitivity. *Dental Clinics of North America* 34, 449-473.

PASHLEY, D.H. (1992) Dentin permeability and dentin sensitivity. *Proceedings of the Finnish Dental Society* 88 (Supplement 1), 31-37.

PASHLEY, D.H., ANDRINGA, H.J. & EICHMILLER, F. (1991) Effects of ferric and aluminum oxalates on dentin permeability. *American Journal of Dentistry* 4, 123-126.

PASHLEY, D.H., DERKSON, G.D., TAO, L., DERKSON, M. & KALATHOOR, S. (1988) The effects of a multi-step dentin bonding system on dentin permeability. *Dental Materials* 4, 60-63.

PASHLEY, D.H. & GALLOWAY, S.E. (1985a) The effects of oxalate treatment on the smear layer of ground surfaces of human dentine. *Archives of Oral Biology* 30, 731-737.

PASHLEY, D.H. & GALLOWAY, S.E. (1985b) Effects of desensitizing solutions on dog dentin permeability. *In vivo*. *Journal of Dental Research* 64, 310 (Abstract no. 1217).

PASHLEY, D.H. & GALLOWAY, S.E. (1985c) The effects of oxalate on smear layers permeability and resistance to acid. *Journal of Dental Research*

64, 238 (Abstract no. 570).

PASHLEY, D.H., GALLOWAY, S.E. & STEWART, F.P. (1984a) Effect of fibrinogen in vivo on dentine permeability in dogs. Archives of Oral Biology 29, 725-728.

PASHLEY, D.H., KEHL, T., PASHLEY, E. & PALMER, P. (1981) Comparison of in vitro and in vivo dog dentin permeability. Journal of Dental Research 60, 763-768.

PASHLEY, D.H., KEPLER, E.E., WILLIAMS, E.C. & O'MEARA, J.A. (1984b) The effect of dentine permeability of time following cavity preparation in dogs. Archives of Oral Biology 29, 65-68.

PASHLEY, D.H., KIM, S., HANKS, C.T., TROWBRIDGE, H.O., et al. (1992a) Consensus report. Proceedings of the Finnish Dental Society 88 (Supplement 1), 571-581.

PASHLEY, D.H., LEIBACH, J.G. & HORNER, J.A. (1987) The effects of burnishing NaF/kaolin/glycerin paste on dentin permeability. Journal of Periodontology 58, 19-23.

PASHLEY, D.H., LIVINGSTON, M.J. & OUTHWAITE, W.C. (1978a) Dentin permeability. Changes produced by iontophoresis. Journal of Dental Research 57, 77-82.

PASHLEY, D.H., LIVINGSTON, M.J., REEDER, O.W. & HORNER, J. (1978b) Effects of the degree of tubule occlusion on the permeability of human dentine in vitro. Archives of Oral Biology 23, 1127-1133.

PASHLEY, D.H., O'MEARA, J.A., KEPLER, E.E., GALLOWAY, S.E., THOMPSON, S.M. & STEWART, F.P. (1984c) Dentin permeability. Effects of desensitizing dentifrices in vitro. Journal of Periodontology 55, 522-525.

PASHLEY, D.H. & PARSONS, G.S. (1987) Pain produced by topical anaesthetic ointment. Endodontics & Dental Traumatology 3, 80-82.

PASHLEY, E., HORNER, J., LIU, M., KIM, S. & PASHLEY, D. (1992b) The effects of CO₂ laser energy on dentin permeability. Journal of Dental Research 71, 162 (Abstract no. 453).

PAWLOWSKA, J. (1956) Znaczeniew Zebolcznictwie. I profilaktyce (Strontium chloride its importance in dentistry and prophylaxis). Czas

Stomatologica 9, 353-361.

PEDEN, J.W. (1977) Dental hypersensitivity. The Journal of the Western Society of Periodontology. Periodontal Abstracts 25, 75-83.

PENNEY, D.A. & KARLSSON, U.L. (1976) Fast desensitization of tooth roots by topically applied SnF_2 and SrCl_2 in dogs. Archives of Oral Biology 21, 339-347.

PERMINDER, K., SHADANI, D.R. & ANTIA, F.E. (1985) A comparative study of the desensitizing effects of dentifrices containing sodium monofluorophosphate and formalin. Journal of the Indian Dental Association 57, 453-458.

PERSON, P., DEMAND, E.E., KOLTUN, L. & SPINDEL, M. (1989) A Microprocessor Temperature-Controlled Air Delivery System for Dentinal Hypersensitivity Testing. Clinical Preventive Dentistry 11, 3-9.

PHANTAMVANIT, P., PRAPAKAMOL, S., ANGCHANPEN, O. & SONGPAISAN, P. (1990) Clinical study of Aluminium Lactate in desensitizing dentifrices. Journal of Dental Research 69, 356 (Abstract no. 1977).

PHILLIPS, R.W. (1973) Skinner's Science of Dental Materials 7th Edition. W.B. Saunders Company, Philadelphia Pp 48 & 467.

PHILLIPS, R.W., JOHNSON, R.J. & PHILLIPS, L.J. (1956) Improved method for measuring the coefficient of thermal conductivity of dental cements. Journal of the American Dental Association 53, 577-583.

POLHAGEN, L. & BRÄNNSTRÖM, M. (1971) The liquid movement in desiccated and rehydrated dentin *in vitro*. Acta Odontologica Scandinavica 29, 95-102.

POLSON, A.M., CATON, J.G., YEAPLE, R.N. & LANDER, H.A. (1980) Histological determination of probe tip penetration into gingival sulcus of humans using an electronic pressure sensitive probe. Journal of Clinical Periodontology 7, 479-488.

POWELL, L.V., GORDON, G.E. & JOHNSON, G.H. (1990) Sensitivity restored of class v abrasion/erosion lesions. Journal of the American Dental Association 121, 694-697.

POWERS, M.M. (1952) The staining of nerve fibres in teeth. Journal of Dental Research 31, 383-392.

- PRAPAKAMOL, S., ANGCHANPEN, O., SONGPAISAN, Y. & PHANTUMVANIT, P. (1991) Al-Lactate desensitizing dentifrice by mechanical, thermal & chemical stimuli. *Journal of Dental Research* 70, 855 (Abstract no. 4).
- PRICE, D.D., McGRATH, P.A., RAFFI, A. & BUCKINGHAM, B. (1983) The validation of visual analogue scales as ratio scale measures for chronic and experimental pain. *Pain* 17, 45-56.
- RAPP, R., AVERY, J.K. & RECTOR, R.A. (1957) A study of the distribution of nerves in human teeth. *Journal of the Canadian Dental Association* 23, 447-453.
- RAPP, R., AVERY, J.K. & STRACHAN, D.S. (1968) Possible role of acetylcholinesterase in neural conduction within the dental pulp. In *Biology of the Dental Pulp Organ*. Finn, S.B. (ed) Alabama, University of Alabama Press Pp 309-325.
- READER, A. & FOREMAN, D.W. (1981a) An ultrastructural qualitative investigation of human intradental innervation. *Journal of Endodontics* 7, 161-168.
- READER, A. & FOREMAN, D.W. (1981b) An ultrastructural quantitative investigation of human intradental innervation *Journal of Endodontics* 7, 493-499.
- REDMALM, G. (1986) Dentifrice abrasivity. The use of laser light for determination of the abrasive properties of different silicas. An *in vitro* study. *Swedish Dental Journal* 10, 243-250.
- REINHART, T.C., KILLOY, W.J., LOVE, J., OVERMAN, P.R. & SAKUMURA, J.S. (1990) The effectiveness of a patient-applied tooth desensitizing gel. A pilot study. *Journal of Clinical Periodontology* 17, 123-127.
- REISSTEIN, J., LUSTMAN, I., HERSHKOVITZ, J. & GEDALIA, I. (1978) Abrasion of enamel and cementum in human teeth due to toothbrushing. Estimated by SEM. *Journal of Dental Research* 57, 42.
- RENTON-HARPER, P. & MIDDA, M. (1992) NdYAG laser treatment of dentinal hypersensitivity. *British Dental Journal* 172, 13-16.
- REYNOLDS, C. (1968) Hypersensitivity. *Oral Health* 58, 295.
- RIPA, L.W. (1989) Clinical studies of high-potency fluoride dentifrices. A review. *Journal of the American Dental Association* 118,

85-91.

ROANE, J.B., FOREMAN, D.W., MELFI, R.C. & MARSHALL, F.J. (1973) An ultrastructure study of dentinal innervation in the adult tooth. *Oral Surgery, Oral Medicine, Oral Pathology* 35, 94-104.

ROBB, N.B. & SMITH, B.G.N. (1992) Influence of toothbrushing practices on toothwear in dental attenders. *Journal of Dental Research* 71, 180 (Abstract no. 598).

ROGERSON, M.J. (1973) The role of a calcium sucrose phosphate-calcium orthophosphate complex in the reduction of dental caries. *Australian Dental Journal* 18, 160-166.

ROSEN, M. & STALLARD, R.E. (1980) Clinical evaluation of Prednisol Plus. *Quintessence International* 11, 107-114.

ROSENTHAL, M.W. (1990) Historic review of the management of tooth hypersensitivity. *Dental Clinics of North America* 34, 403-427.

ROSS, M.R. (1961) Hypersensitive teeth. Effect of strontium chloride in a compatible dentifrice. *Journal of Periodontology* 32, 49-53.

RUGG-GUNN, A.J. & MacGREGOR, I.D.M. (1978) A survey of toothbrushing behaviour in children and young adults. *Journal of Periodontal Research* 13, 382-389.

SALVATO, A., CLARK, G. & CURRO, F.A. (1989) Clinical efficacy of Sensodyne-F as a desensitizing dentifrice. *Journal of Dental Research* 68, 971 (Abstract no. 839).

SALVATO, A., TROULLOS E.S., CURRO, F.A., MEROLA, M.R. & GINGOLD, J. (1990) The Effectiveness of Ferric Oxalate (FO) in relieving Dentinal Hypersensitivity. *Journal of Dental Research* 69 (Special Issue), 169 (Abstract no. 482).

SAMARA-WICKRAMA, D.Y.D., SILVERSTONE, L.M. & JOHNSON, N.W. (1972) Effect of calcifying fluids on human dentin *in vitro*. *Journal of Dental Research* 51, 1268-1269.

SANDOVAL, V.A., COOLEY, R.L., & BARNWELL, S.E. (1989) Evaluation of potassium oxalate as a cavity liner. *Journal of Prosthetic Dentistry* 62, 283-287.

SANGNES, G. (1976) Traumatization of teeth and gingiva related to

habitual tooth cleaning procedures. *Journal of Clinical Periodontology* 3, 94-103.

SAUSEN, R.E. (1955) Electrophoresis in dentin with radio-active calcium. *Journal of Dental Research* 34, 12-19.

SCHAEFFER, M.L., BIXLER, D. & YU, P-L. (1971) The effectiveness of iontophoresis in reducing cervical hypersensitivity. *Journal of Periodontology* 42, 695-700.

SCHAFFNER, M., SUTER, P., HOTZ, P. & LUSI, A. (1988) Epidemiology of dental erosion and toothbrush abrasion. *Journal of Dental Research* 67, 388 (Abstract no. 2205).

SCHERMAN, A. & JACOBSEN, P.L. (1992) Managing dentin hypersensitivity: what treatment to recommend to patients. *Journal of the American Dental Association* 123, 57-61.

SCHLUGER, S., YUDELIS, R.A. & PAGE, R.C. (1977) *Periodontal Disease*. Lea & Febiger, Philadelphia, Pp 769-770.

SCOTT, D. (1966) Excitation of the dentinal receptor in the tooth of the cat. In: *Touch, Heat and Pain*, De Reuck, A.V.S. & Knight, J. (eds) Churchill, London Pp 261-273.

SCOTT, D. (1972) The arousal and suppression of pain in the teeth. *International Dental Journal* 22, 20-32.

SCOTT, D. (1974) Physiologic considerations in the arousal of pain in teeth. Structure and function of existing mechanisms and a hypothesis of their transducer action. In: Chasens, A.I. & Caslick, R.S. (eds). *Workshop on Mechanisms and Pain and Sensitivity in the Teeth and Supporting Tissues*, Fairleigh Dickinson University, School of Medicine & American Academy of Oral Medicine, 12-13 November Pp 24-23.

SCOTT, D. (1982) The physiologic origins of dental pain. *Compendium of Continuing Education in Dentistry. Supplement* 3, 92, 126.

SCOTT, D. & STEWART, G.G. (1965) Excitation of the dentinal receptor of the cat by heat and chemical agents. *Oral Surgery, Oral Medicine, Oral Pathology* 20, 784-794.

SCOTT, D. & TEMPEL, T.R. (1965) Neurophysiological response of single receptor units in the tooth of the cat. *Journal of Dental Research* 44,

20-27.

SCOTT, H.M. (1962) Reduction of sensitivity by electrophoresis. *Journal of Dentistry in Children* 19, 225-241.

SCOTT, J. & HUSKISSON, E.C. (1976) Graphic representation of pain. *Pain* 2, 175-184.

SELTZER, S. (1978) *Pain Control in Dentistry*. Philadelphia, J.B. Lippincott Company Pp 105-136.

SELTZER, S. & BENDER, I.B. (1975a) The nerve supply of the pulp and pain perception. In. *The Dental Pulp*, 2nd Edition. Philadelphia, J.B. Lippincott Company Pp 131-151.

SELTZER, S. & BENDER, I.B. (1975b) Desensitizing and remineralizing agents. In. *The Dental Pulp*, 2nd edition. Philadelphia, J.B. Lippincott Company, Pp 202-210.

SELTZER, S. & BENDER, I.B. (1990) Glucocorticoids. In. *The Dental Pulp*. 3rd Edition. Philadelphia, J.B. Lippincott Company, p 222.

SELTZER, S., BENDER, I.B. & ZIONTZ, M. (1963) The diagnosis of pulp inflammation, correlations between diagnosis data and actual histological findings in the pulp. *Oral Surgery, Oral Medicine, Oral Pathology* 16, 846-871.

SENA, F.J. (1990) Dentinal permeability in assessing therapeutic agents. *Dental Clinics of North America* 34, 475-490.

SEO, M.S. & PARK, D.S. (1991) The effectiveness of potassium oxalate and sodium fluoride on the reduction of dentinal sensitivity. *Journal of Dental Research* 70, 818 (Abstract no. 0-91).

SEYMOUR, R.A. ((1982) The use of pain scales in assessing the efficacy of analgesics in post-operative dental pain. *European Journal of Clinical Pharmacology* 23, 441-444.

SHAPIRO, W.B., KASLICK, R.S. & CHASENS, A.I. (1970a) The effect of a strontium chloride toothpaste on root hypersensitivity in a controlled clinical study. *Journal of Periodontology* 41, 702-703.

SHAPIRO, W.B., KASLICK, R.S. CHASENS, A.I. & WEINSTEIN (1970b) Controlled clinical comparison between a strontium chloride and a sodium monofluorophosphate toothpaste in diminishing root hyper-

sensitivity. *Journal of Periodontology* 41, 523-525.

SHULMAN, L.B., GEDALIA, I. & FEINGOLD, R.M. (1973) Fluoride concentration in root surfaces and alveolar bone of fluoride-immersed monkey incisors three weeks after replantation. *Journal of Dental Research* 52, 1314-1316.

SHULMAN, L.B., KALIS, P. & GOLDBERGER, P. (1968) Fluoride inhibition of tooth-replant root resorption in cebus monkeys. *Journal of Oral Therapeutics & Pharmacology* 4, 331-337.

SIDI, A.D., WILSON, R.F. & ASHLEY, F.P. (1991) Efficacy of a new toothpaste formulation in reducing dentine hypersensitivity. *Journal of Dental Research* 70, 682 (Abstract no. 110).

SIEW, C., GRUNINGER, S.E., O'YOUNG, A., TS'AO, N.K. & BOWEN, R.L. (1984) Biological safety evaluation of a novel dentin bonding system. *Journal of Dental Research* 63, 313 (Abstract no. 1277).

SIGAL, M.J., AUBIN, J.E., TEN CATE, A.R. & PITATU, S. (1984a) The odontoblast process extends to the dentinoenamel junction: an immunocytochemical study of rat dentine. *Journal of Histochemistry & Cytochemistry* 32, 872-877.

SIGAL, M.J., PITATU, S., AUBIN, J.E. & TEN CATE, A.R. (1984b) A combined scanning electron microscopy and immunofluorescence study demonstrating that the odontoblast process extends to the dentinoenamel junction in human teeth. *Anatomical Record* 210, 453-462.

SIGAL, M.J., AUBIN, J.E. & TEN CATE, A.R. (1985) An immunocytochemical study of the human odontoblast process using antibodies against tubulin, actin and vimentin. *Journal of Dental Research* 64, 1348-1355.

SILNESS, J. & LÖE, H. (1964) Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. *Acta Odontologica Scandinavica* 22, 121-135.

SILVERMAN, G. (1985) The sensitivity-reducing effect of brushing with a potassium nitrate-sodium monofluorophosphate dentifrice. *Compendium of Continuing Education in Dentistry* 6, 131-136.

SILVERMAN, G. (1986) The effectiveness of two dentifrices in relieving dental hypersensitivity. *Journal of Dental Research* 65 (Special Issue),

297 (Abstract no. 1150).

SILVERMAN, G., GINGOLD, J. & CLARK, G.E. (1988) The effectiveness of KNO_3 and NaMFP dentifrices in reducing dentinal hypersensitivity.

Journal of Dental Research 67, 247 (Abstract no. 1076).

SINGH, V.B., GOVILA, C.P. & MATHUR, R.M. (1984) Efficacy of desensitizing agents in dentifrices for hypersensitive teeth. Journal of the Indian Dental Association 56, 305-309.

SKURNIK, H. (1963) Control of dental hypersensitivity. Preliminary report on a strontium-containing dentifrice. Journal of Periodontology 34, 183-185.

SLAYTON, R.L., WEFEL, J.S. & SWIFT, E.J. (1992) Effects of laser irradiation on permeability of human root dentin. Journal of Dental Research 71, 218 (Abstract no. 903).

SMITH, B.A. & ASH, M.M. (1964a) Evaluation of a desensitizing dentifrice. Journal of the American Dental Association 68, 639- 647.

SMITH, B.A. & ASH, M.M. (1964b) A study of a desensitizing dentifrice and cervical hypersensitivity. Journal of Periodontology 35, 222-231.

SMITH, B.A., HANSSON, R.E., CAFFESSE, R.G. & BYE, F.L. (1988) Evaluation of dipotassium oxalate in treatment of root hypersensitivity. Journal of Dental Research 67, 329 (Abstract no. 1733).

SQUILLARO, R., WELSH, C.A., CHILTON, N.W. & YANKELL, S.L. (1981) Clinical trial of a NaF gel on dental cervical hypersensitivity. Journal of Dental Research 60 (Special Issue A) 461 (Abstract no. 608).

STANLEY, H.R. (1974) Dentin permeability and sensitivity. In: Chasens, A.I. & Kaslick, R.S. (eds) Workshop on Mechanisms of Pain and Sensitivity in the Teeth and Supporting Tissues. Fairleigh Dickinson University, School of Medicine & American Academy of Oral Medicine, 12-13 November, Pp 34-45.

STANLEY, H.R., SWERDLOW, H. & DRISCOLL, E.J. (1965) Minimizing pulpal reactions with prednisolone therapy. The effects of the components of a steroid formula. Journal of Oral Therapeutics and Pharmacology 2, 1-8.

STANLEY, H.R., GOING, R.E. & CHAUNCEY, H.H. (1985a) Human pulp response

to acid pretreatment of dentin and to composite restoration. Journal of the American Dental Association 91, 817-825.

STANLEY, H.R., BOWEN, R.L. & COBB, E.N. (1985b) Pulp response to experimental treatments of dentin for bonding composites. Journal of Dental Research 64, 222 (Abstract no. 423).

STARR, C.B., MAYHEW, R.B. & PIERSON, W.P. (1989) The effect of hypnosis in the treatment of dentin sensitivity. General Dentistry Jan/Feb 13-15.

STARK, M.M., KEMPLER, D., PELZNER, R.B., ROSENFELD, J., LEUNG, R.L. & MINTATOS, S. (1977) Rationalization of electric pulp-testing methods. Oral Surgery, Oral Medicine, Oral Pathology 43, 598-606.

STARK, M.M. & PELGNER, R. (1982) Measurement of dentinal hyper-sensitivity. Compendium of Continuing Education in Dentistry Supplement 3, 110-112.

STEPHAN, R.M. (1937) Correlation of clinical tests with microscopic pathology of dental pulp. Journal of Dental Research 16, 267-278.

STOUT, W.C. (1955) Sodium silicofluoride as a desensitizing agent. Journal of Periodontology 26, 208-210.

STOOKEY, G.K. & MUHLER, J.C. (1968) Laboratory studies concerning the enamel and dentin abrasion properties of common dentifrices polishing agents. Journal of Dental Research 47, 524-532.

STOWELL, E.C., TAYLER, J.B. & WAINWRIGHT, W.W. (1961) Ion penetration through teeth as influenced by an electrostatic field. Journal of Dental Research 40, 739-740.

SUTHERLAND, L., JENSEN, M.E., HAWKINS, B.F. & LAINSON, P.A. (1989) Root caries inhibition using dentin bonding resin and fluoridated varnishes. Journal of Dental Research 68, 279 (Abstract no. 786).

SWERDLOW, H., STANLEY, H.R. & SAYEGH, F.S. (1965) Minimizing pulpal reactions with prednisolone therapy. Journal of Oral Therapeutics and Pharmacology 1, 593-601.

SZABÓ, J., SZABÓ, I. & TROMBITÁS, K. (1985) Scanning electron microscopy of the walls of tubules in human coronal dentine. Archives of Oral Biology 30, 705-710.

- TAKAHASI, A. (1986)** An in vitro study in the effects of various agents on dentin permeability. Bulletin of Kanagawa Dental College 14, 5-15.
- TAKAHASHI, K. (1990)** Concluding remarks. In Dynamic Aspects of Dental Pulp, Molecular Biology, Pharmacology and Pathophysiology. Inoki, R., Kudo, T. & Olgart, L.M. (eds) Chapman & Hall, London Pp 131-132.
- TAL, M., ORON, M., GEDALIA, I. & EHRLICH, J. (1976)** X-ray diffraction and Scanning Electron Microscope investigations of fluoride-treated dentin in man. Archives of Oral Biology 21, 285-290.
- TALEGHANI, M. & LEINFELDER, K. (1991)** Two year clinical evaluation of Tenure dentin bonding agent. Journal of Dental Research 70, 457 (Abstract no. 1530).
- TALHI, F.S., DAVIES, E.H. & NALLY, F.F. (1985)** Nerve recovery after cryotherapy for paroxysmal trigeminal neuralgia. Journal of Dental Research 64, 694 (Abstract no. 286).
- TARBET, W.J., SILVERMAN, G., STOLMAN, J.M. & FRATARCANGELO, P.A. (1979)** An evaluation of two methods for the quantification of dentinal hypersensitivity. Journal of the American Dental Association 98, 914-918.
- TARBET, W.J., SILVERMAN, G., STOLMAN, J.M. & FRATARCANGELO, P.A. (1980)** Clinical evaluation of a new treatment for dentinal hypersensitivity. Journal of Periodontology 51, 535-540.
- TARBET, W.J., SILVERMAN, G., FRATARCANGELO, P.A. & KANAPKA, J.A. (1982)** Home treatment for dentinal hypersensitivity. A comparative study. Journal of the American Dental Association 105, 227-230.
- TAVARES, M., DEPAOLA, P. & SOPARKAR, P. (1992)** Use of a fluoride releasing resin for reducing cervical sensitivity. Journal of Dental Research 71, 311 (Abstract no. 1640).
- TEN-CATE, A.R., ANDERSON, R.D. & SIGAL, M. (1985)** The functional role of the odontoblast-its response to cavity preparation. In Current Topics in Oral Biology. Lisney, S.J.W. & Matthews, B. (eds) Bristol, University of Bristol press Pp 159-168.
- TEN-CATE, A.R. & SHELTON, L. (1966)** Cholinesterase activity in human teeth. Archives of Oral Biology 11, 423-428.
- TERKLA, L.G., BROWN, A.C., HAINISCH, A.P. & MITCHEM, J.C. (1987)**

Testing sealing properties of restorative materials against moist dentin. *Journal of Dental Research* 66, 268 (Abstract no. 1292).

THRASH, W.J., DORMAN, H.L. & SMITH F.D. (1983). A method to measure pain associated with hypersensitive dentin. *Journal of Periodontology* 54, 160-162.

THRASH, W.J., JONES, D.L. & DODDS, M.W.J. (1992) Effects of a solution containing 1.09% NaF, .40% SnF₂ & .14% hydrogen fluoride on dentinal pain. *Journal of Dental Research* 71, 158 (Abstract no. 418).

TJAN, A.H.L. & CHAN, C.A.C. (1987) Effects of resin-compatible cavity varnishes on the polymerization of visible light-cured composites. *Journal of Prosthetic Dentistry* 58, 559-563.

TJAN, A.H.L., GRANT, B.E. & NEMETZ, H. (1987) The efficacy of resin-compatible cavity varnishes in reducing dentin permeability to free monomer. *Journal of Prosthetic Dentistry* 57, 179-185.

TOTO, P.D., STAFFILENO, H. & GARGIULO, A.W. (1958) A clinical evaluation of a desensitizing toothpaste. *Journal of Periodontology* 29, 192-194.

TOUYZ, L.Z.G. (1983) Fruit induced sensitivity at cervical margins. *Journal of the Dental Association of South Africa* 38, 199-200.

TRONSTAD, L. & LANGE LAND, K. (1971a) Histochemical observation on human dentine exposed by attrition. *Scandinavian Journal of Dental Research* 79, 151-159.

TRONSTAD, L. & LANGE LAND, K. (1971b) Electron microscopy of human dentine exposed by attrition. *Scandinavian Journal of Dental Research* 79, 160-171.

TROWBRIDGE, H.O. (1982) Mechanisms and control of pain in dentin and pulp. In. Rowe, N.H. (ed) *Proceedings of Symposium on Dental Pulp: Reactions to Restorative Materials in the Presence or Absence of Infection*. University of Michigan Pp 1-20.

TROWBRIDGE, H.O. (1985) Mechanisms of pain induction in hypersensitive teeth. In. Rowe, N.H. (ed). *Proceedings of symposium on hypersensitive teeth. Origin and management*. University of Michigan, Pp 1-19.

TROWBRIDGE, H.O. (1986) Review of dental pain. *Histology and*

physiology. *Journal of Endodontics* 12, 445-452.

TROWBRIDGE, H.O. (1991) Tooth sensitivity: Origin, diagnosis and treatment. *Journal of Dental Research* 70, 451 (Abstract no. 1482).

TROWBRIDGE, H.O., EDWALL, L. & PANOPOULOS, P. (1982) Effect of zinc-oxide eugenol and calcium hydroxide on intradental nerve activity. *Journal of Endodontics* 8, 403-406.

TROWBRIDGE, H.O., FRANKS, M., KOROSTOFF, E. & EMLING, R. (1980) Sensory response to thermal stimulation in human teeth. *Journal of Endodontics* 6, 405-412.

TROWBRIDGE, H.O. & SILVER, D.R. (1990) A review of current approaches to in-office management of tooth hypersensitivity. *Dental Clinics of North America* 34, 561-581.

TURKER, M.N. (1975) A method for studying the peripheral mediators of the dental pain induced by electrical stimulation. *Archives of International Physiology and Biochemistry* 17, 1059.

UCHIDA, T., KIKUCHI, T. & AZUMA, T. (1975) Clinical investigation of the toothpaste containing aluminium lactate and aluminium fluoride. *Journal of Hiroshima University Dental Society* 7, 60-66.

UCHIDA, A., WAKANO, Y., FUKUYAMA, O., MIKI, T., IWAYAMA, Y. & OKADA, H. (1980) Controlled clinical evaluation of a 10% strontium chloride dentifrice in treatment of dentin hypersensitivity following periodontal surgery. *Journal of Periodontology* 51, 578-581.

VEZIN, J.C., BOUNOURE, G.M., MONDAIN, J. & POITOU, P. (1985) Mise en évidence d'une diminution de la solubilité de l'émail après traitement au fluorohydrate de nicométhanol. *Pharmaceutica Acta Helvetiae* 60, 145-148.

VIRTANEN, A., NÄRHI, M., HUOPANIEMI, T. & HIRVONEN, T. (1983) Thresholds of intradental A- and C- fibres in the cat to electrical current pulses of different duration. *Acta Physiologica Scandinavica* 119, 393-398.

VONGSAVAN, N. & MATTHEWS, B. (1991) The permeability of cat dentine *in vivo* and *in vitro*. *Archives of Oral Biology* 36, 641-646.

VONGSAVAN, N. & MATTHEWS, B. (1992a) Hydrodynamic effects in human

dentine in vitro. Journal of Dental Research 71, 742 (Abstract no. 1814).

VONGSAVAN, N. & MATTHEWS, B. (1992b) Fluid flow through cat dentine in vivo. Archives of Oral Biology 37, 175-185.

VOTH, E.D., PHILLIPS, R.W. & SWARTZ, M.L. (1966) Thermal diffusion through amalgam and various liners. Journal of Dental Research 45, 1184-1190.

WAKABAYASHI, H. & MATSUMOTO, K. (1988) Treatment of dentine hypersensitivity by GaAlAs soft laser irradiation. Journal of Dental Research 67, 182 (Abstract no. 554).

WALLACE, J.A. & BISSADA, N.F. (1990) Pulpal and root sensitivity rated to periodontal therapy. Oral Surgery, Oral Medicine, Oral Pathology 69, 743-747.

WALTON, R.E., LEONARD, L.A., SHARAWY, M. & GANGAROSA, L.P. (1979) Effects on pulp and dentin of iontophoresis of sodium fluoride on exposed roots in dogs. Oral Surgery, Oral Medicine, Oral Pathology 48, 545-557.

WALTON, J.G., THOMPSON, J.W. & SEYMOUR, R.A. (1989) Textbook of Dental Pharmacology and Therapeutics., Oxford University Press p 240.

WARFINGE, J., DAHLEN, G. & BERGENHOLTZ, G. (1985) Dental pulp response to bacterial cell wall material. Journal of Dental Research 64, 1046-1050.

WASSERMAN, F. (1939) The innervation of the teeth. Journal of the American Dental Association 26, 1097-1109.

WEI, S.H.Y., LAINSON, P.A., HENDERSON, W. & WOLFSON, S.H. (1980) Evaluation of dentifrices for the relief of hypersensitive tooth surfaces. Quintessence International 11, 67-73.

WEINE, F.S. (1982) Endodontic therapy. 3rd edition. C.V. Mosby, Saint Louis. Pp 56-59.

WELLING, A.W. (1940) A critical review of recent histology and entrance of nerves into dentine. Proceedings of the Royal Society of Medicine 33, 563-576.

WHITE, J.M., GOODIS, H.E., MARROQUIN, E., MARSHALL, S.J. & MARSHALL,

- G.W. (1992) Physical modification threshold of dentin with Nd & Ho:YAG lasers. *Journal of Dental Research* 71, 218 (Abstract no. 902).
- WHITE, J.M., GOODIS, H.E. & ROSE, C.M. (1990) Effect of Nd:YAG laser treatments on hydraulic conductance of dentin. *Journal of Dental Research* 69, 169 (Abstract no. 481).
- WILDER-SMITH, P. (1988) The soft laser. Therapeutic tool or popular placebo? *Oral Surgery, Oral Medicine, Oral Pathology* 66, 654-658.
- WINTER, H.F., BISHOP, J.G. & DORMAN, H.L. (1963) Transmembrane potential of odontoblasts. *Journal of Dental Research* 42, 594-598.
- WOODROW, K.M., FRIEDMAN, G.D., SIEGELAUB, A.B. & COLLEN, M.F. (1972) Pain tolerance: differences according to age, sex and race. *Psychosomatic Medicine* 34, 548-556.
- WOOFER, C. (1969) The prevalence and etiology of gingival recession. *Periodontal Abstracts* 17, 45-50.
- WYCOFF, S.J. (1982) Current treatment for dentinal hypersensitivity. *Compendium of Continuing Education in Dentistry Supplement* 3, 113-115.
- XU, G.Q. (1980) Maxilla facial nerve desensitisation. In: *Textbook of oral medicine*, Sichuan Medical College (ed), Peoples Sanitarians press, Beijing, China Pp 341-348.
- YANKELL, S.L. (1982) Current treatment for dentinal hypersensitivity. *Compendium for Continuing Education in Dentistry Supplement* 3, 115-117.
- YEH, K., DANGLER, L., SENA, F., ZIENTEK, L. DENOOYER, M. & WEDDLE, L. (1990) Use of ferric oxalate to reduce dentin hypersensitivity. *Journal of Dental Research* 69, 168 (Abstract no. 479).
- YOSHIYAMA, M., MASADA, J., UCHIDA, A. & ISHIDA, H. (1989) Scanning electron microscopic characterization of sensitive vs. insensitive human radicular dentin. *Journal of Dental Research* 68, 1498-1502.
- YOSHIYAMA, M., NOIRI, Y., OZAKI, K., UCHIDA, A., ISHIKAWA, Y. & ISHIDA, H. (1990) Transmission electron microscopic characterization of hypersensitive human radicular dentin. *Journal of Dental Research* 69, 1293-1297.
- YOSHIYAMA, M., OZAKI, K. & EBISU, S. (1992) Morphological characterization of hypersensitive human radicular dentin and the

effect of a light-curing resin liner on tubular occlusion. Proceedings of the Finnish Dental Society 88 (Supplement 1), 337-344.

ZADOK, J., GEDALIA, I., WEIMAN, J. & DAPHNI, I. (1976) Fluoride uptake by root dentin after immersion in 2% NaF solution with iontophoresis. Journal of Dental Research 55, 310-314.

ZAKRZEWSKA, J.M. & FEINMANN, C. (1990) A standard way to measure pain and psychological morbidity in dental practice. British Dental Journal 169, 337-339.

ZIGMOND, A.S. & SNAITH, R.P. (1983) The Hospital Anxiety and Depression Scale. Acta Psychologica Scandinavica 67, 361-370.

ZINNER, D.D., DUANY, L.F. & LUTZ, H.J. (1977) A new desensitizing dentifrice. Preliminary report. Journal of the American Dental Association 95, 982-985.

APPENDICESPUBLICATIONS

- DAVIES, E.H., GILLAM, D.G., NEWMAN, H.N., BULMAN, J.S. & MILLAR, I. (1993) Development and laboratory evaluation of the biomat thermal probe (BTP). Journal of Dental Research 72, 329 (Abstract no. 1803).
- GILLAM, D.G., BULMAN, J.S. & NEWMAN, H.N. (1992) Quantification of pain in cervical dentinal sensitivity (CDS) studies. Journal of Dental Research 71, 628 (Abstract no. 902).
- GILLAM, D.G., BULMAN, J.S. & NEWMAN, H.N. (1993) Use of word descriptors in cervical dentinal sensitivity (CDS) studies. Journal of Dental Research 72, 740 (Abstract no. 430).
- GILLAM, D.G., DAVIES, E.H., NEWMAN, H.N. & BULMAN, J.S. (1993) Clinical evaluation of the biomat thermal probe (BTP). Hypersensitivity dentine. Biological basis of therapy symposium. Callaway gardens, Georgia. 14-16th March, p23, Poster no. 03.
- GILLAM, D.G. & NEWMAN, H.N. (1990) Iontophoresis: A review. Journal of Western Society of Periodontology and Periodontal Abstracts 38, 129-133.
- GILLAM, D. & NEWMAN, H. (1992) Treating sensitivity. Oral Hygiene. A Chemist & Druggist supplement July 25th, Pp 5-6.
- GILLAM, D.G. & NEWMAN, H.N. (1993) Assessment of pain in cervical dentinal sensitivity studies - A review. Journal of Clinical Periodontology 20, 383-394).
- GILLAM, D.G., NEWMAN, H.N. & BULMAN, J.S. (1992) Effect of SCH dentifrices on plaque and gingivae. Journal of Dental Research 71, 584 (Abstract no. 547).
- GILLAM, D.G., NEWMAN, H.N. & BULMAN, J.S. (1992) The effect of strontium chloride hexahydrate dentifrices on plaque accumulation and gingival condition. Journal of Clinical Periodontology 19, 737-740.
- GILLAM, D.G., NEWMAN, H.N., BULMAN, J.S. & DAVIES, E.H. (1992) Dentifrice abrasivity and cervical dentinal hypersensitivity. Results 12 weeks following cessation of 8 weeks' supervised use. Journal of

Periodontology 63, 7-12.

GILLAM, D.G., NEWMAN, H.N., DAVIES, E.H. & BULMAN, J.S. (1991)

Dentifrice abrasivity and cervical dentinal hypersensitivity. Journal of Dental Research 70, 694 (Abstract no. 205).

GILLAM, D.G. NEWMAN, H.N., DAVIES, E.H. & BULMAN, J.S. (1992) Clinical

efficacy of a low abrasive dentifrice for the relief of cervical dentinal hypersensitivity. Journal of Clinical Periodontology 19, 197-201.

Dentifrice Abrasivity and Cervical Dental Hypersensitivity. Results 12 Weeks Following Cessation of 8 Weeks' Supervised Use

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FOLLOWING AN 8-WEEK CONTROLLED INVESTIGATION of 2 strontium chloride hexahydrate dentifrices (SCH) of differing abrasivity, 2 groups of 20 subjects each, with cervical dental hypersensitivity, were re-examined at 20 weeks; that is, 12 weeks after the active period. The examination procedures were conducted in the same manner as in the main clinical trial. Sensitivity levels were assessed by 2 instrument methods: tactile (Yeaple probe), and cold air (dental air syringe), and by subjective perception of pain by means of a Visual Analogue Scale. The results from these methods of assessment demonstrated that 12 weeks following the cessation of 8 weeks' controlled use of standard and low abrasive SCH dentifrices, sensitivity levels reversed only slightly in both groups and, overall, sensitivity remained significantly lower than at baseline. The abrasivity of the dentifrice did not affect the desensitization activity. *J Periodontol* 1992; 63:7-12.

Key Words: Dentin, sensitive; dentifrices; strontium chloride; pain perception.

Strontium chloride hexahydrate (SCH) has been widely used in a dentifrice form for the treatment of cervical dental hypersensitivity (CDH).¹⁻⁷ Concern, however, has been expressed regarding the lack of information about quantification of the test stimuli under suitably controlled conditions, as well as to the absence of an objective method for evaluating the dentifrice's effect in reducing CDH.⁸ These deficiencies were addressed in a 12-week, double-blind, parallel comparative (placebo) study,⁹ in which levels of hypersensitivity in affected teeth were assessed by 3 methods: thermally controlled cold air stimulus; tactile stimulus with an electronic pressure-sensitive probe; and subjective response. The authors concluded that the results from all 3 methods of assessment indicated that SCH was significantly more effective than a placebo in reducing CDH.

Some have questioned the effectiveness of SCH.^{10,11} Others have suggested that reductions in sensitivity previously attributed to the active ingredient in desensitizing dentifrices may, in fact, be attributable to the abrasive components of the dentifrice; notably, silica, contributing to the formation of a smear layer which effectively blocks the exposed dental tubule orifices.¹²⁻¹⁸

Recent studies^{11,19-24} have utilized a low abrasive component in such dentifrices with varying results. Some^{11,23,24}

reported that a silica-based product containing strontium acetate and fluoride (SrAc₂F) was more effective than SCH dentifrice containing the abrasive diatomaceous earth. Gillam et al.,²⁵ however, showed that SCH dentifrices containing the abrasive diatomaceous earth or precipitated silica were equally effective in reducing CDH. Few clinical studies based on the Council on Dental Therapeutics recommendations⁸ for objective as well as subjective methods for evaluating dentifrice effects in reducing CDH have reported any follow-up data following cessation of dentifrice use. The purpose of this present study was to provide such data, based on 3 accepted methods of assessment, following cessation of controlled SCH dentifrice use.

MATERIALS AND METHODS

During the original 8-week randomized double-blind 2-way comparative parallel study of 40 patients, a non-commercially available SCH dentifrice with a silica-based abrasive was compared with a commercially available SCH dentifrice containing the abrasive diatomaceous earth.⁸ Both dentifrices were closely matched with respect to taste, color, consistency, and appearance. All 40 patients returned to be re-examined at the 20-week point (Table 1). During the follow-up examination, the assessment procedures were as in the original study²⁵ and as summarized below.

Selection of subjects was restricted to those with hyper-

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Table 1. Patient Data

	Number	Silica-Based Group Mean Age (SD)	Number	Diatomaceous Earth Group Mean Age (SD)	Number	Total Mean Age (SD)
Female	13	42.6 (11.38)	12	43.9 (8.24)	25	43.2 (9.81)
Male	7	40.3 (4.88)	8	43.8 (3.84)	15	42.1 (4.57)
Mean	20	41.8 (9.52)	20	43.8 (6.69)	40	42.8 (8.18)

sensitivity accompanied by cervical erosion, abrasion, and/or gingival recession on at least one tooth for tactile stimulus and two for cold air stimulus on teeth anterior to the second molar. Subjects were included who, during baseline examination, experienced sensitivity to a tactile force of 10 to 50 gm and recorded a Visual Analogue Scale (VAS) of 3 to 8 cm following application of a cold air stimulus. All had at least one tooth sensitive to the Yeaple probe and two to the dental air syringe, although not necessarily the same teeth.

Subjects with a history of gingival surgery within the previous 6 months were excluded, as were teeth with suspected pulpitis, caries or cracked enamel, defective restorations, and those used as denture abutments. Subjects with Gingival Index²⁶ ≥ 1 for the gingiva of prospective sensitive and adjacent teeth, or ≥ 2 for non-study teeth were also excluded. Subjects using a desensitizing dentifrice agreed to refrain from using same for at least 2 months prior to the trial.

Subjects were examined for baseline sensitivity using both tactile and cold air stimuli. Sensitive teeth were initially detected with a #6 straight probe cervically on each tooth anterior to the second molar. Ten minutes later the investigator assessed the tooth response to cold air using the standard dental air syringe at 40 to 65 p.s.i. at a temperature of 19°C.

Procedure for Measuring Hypersensitivity

Tactile Method (Yeaple Probe - Modified)

The Yeaple probe⁸ was modified to accept a tip with rounded end 0.30 to 0.35 mm diameter (Williams 14W). The probe is designed to deliver a pre-set force when the tip is applied perpendicular to the cervical labial surface.²⁷ Application of the incremental probe force was continued to the point at which discomfort was just felt and the force setting noted. The maximum force applied was 70 gm. If, following the 2 baseline measurements, the subject did not perceive any discomfort at that force, a score of 70 was recorded. The subject was also asked to rate the perception of sensitivity experienced of tactile probe application by placing a mark on a 10 cm line on a "Tactile Sensitivity Scale Form." The distance of the mark from "no pain" end provided an estimate of pain perceived by the subject and constituted a Tactile VAS Score.

Cold Air (Thermal Method)

Ten minutes later, response was assessed to a 1-second application from a standard dental unit syringe at 40 to 65 p.s.i. at a temperature of 19°C, and directed perpendicular to the exposed root surface after isolating the test tooth. Using the principle of VAS Scores (0-10) air pain intensity was indicated by the subject placing a mark on a 10 cm line on a "Subject Air Sensitivity Score Form." The distance of the mark from the "no pain" end provided an estimate of pain perceived by the subject and constituted an air sensitivity score.

Subjective Reporting of Pain-Baseline

Subjects were asked to rate their perception of sensitivity to hot/cold food and drink, air, toothbrushing, and sweet and sour food by placing a mark on a 10 cm line. The distance of the mark from the "no pain" end provided an estimate of the overall severity of pain perceived by the subject. After approximately 1 week, a second baseline determination was made repeating the above procedure.

Data Analysis

All data were tested for normality by plotting in ascending order of magnitude against the corresponding normal scores. All proved to be normally distributed with the exception of Tactile Force, which was then normalized by means of logarithmic transformation. Data analysis was complicated by the fact that, since the readings were time-dependent, it was not possible to undertake a straightforward multiple regression or analysis of variance. To avoid this, the 4 main sources of data, Tactile Force, Tactile VAS, Cold Air Sensitivity, and Overall Sensitivity VAS, were analyzed independently using the following procedure:

1. Inter-group and within-group comparisons of change in response from baseline to 20 weeks. If this within-group test proved significant then,
2. Inter-group comparison of the rate of change within the study time period,
3. Inter-group comparison of the overall level of response within the study time period.

Of these, 1) was achieved by comparing the differences between group mean scores at baseline and at 20 weeks using firstly a paired *t* test (19 degrees of freedom) to see if there was a significant difference within each group, and secondly an unpaired *t* test (38 degrees of freedom) to measure any relative differences between groups.

Analysis of 2) was accomplished by calculating a

Table 2. Summary of 8-Week Data Response to Tactile Stimulus (Yeaple probe-gm)

	Silica-Based Group (20)	Diatomaceous Earth Group (20)
Log₁₀ Transformed Data		
Baseline mean (S.E.)	1.142 (0.030)	1.132 (0.035)
8-Week Mean (S.E.)	1.399 (0.057)	1.413 (0.061)
Paired <i>t</i> test for ratio (19 d.f.) <i>t</i> :	4.36 *	5.43 *
95% C.I. for true ratio (Baseline) (8 Weeks)	0.42 to 0.74	0.41 to 0.67
This indicates that the 8-week reading would generally be expected to be greater than the baseline reading by a factor (for the silica group) of between 1.35 (= 1/0.74) and 2.38 (= 1/0.42).		
Intergroup comparison (unpaired <i>t</i>) <i>t</i> = 0.284 (38 df) NS.		
Subjective Response to Tactile Stimulus - Visual Analogue Scale		
Baseline mean (S.E.)	3.51 (0.356)	3.46 (0.401)
8-Week mean (S.E.)	1.88 (0.351)	1.78 (0.285)
Paired <i>t</i> test for difference (19 d.f.) <i>t</i>	4.28*	4.63*
95% C.I. for true difference (B-8 week)	0.83 to 2.42	0.92 to 2.44
Intergroup comparison (unpaired <i>t</i>) <i>t</i> = 0.107 (38 df) NS.		
Subjective Response to Cold Stimulus. Dental Air Syringe - Visual Analogue Scale (cm)		
Baseline mean (S.E.)	5.28 (0.284)	5.11 (0.263)
8-Week mean (S.E.)	2.73 (0.506)	2.85 (0.573)
Paired <i>t</i> test for difference (19 d.f.) <i>t</i> :	4.69*	5.04*
95% C.I. for true difference	1.41 to 3.70	1.32 to 3.21
Intergroup comparison (unpaired <i>t</i>) <i>t</i> = 0.410 (38 df) NS.		
Visual Analogue Scale (cm) for Overall Perceived Discomfort to Everyday Stimuli		
Baseline mean (S.E.)	4.15 (0.429)	4.44 (0.453)
8-Week mean (S.E.)	1.94 (0.433)	2.16 (0.439)
Paired <i>t</i> test for difference (19 d.f.) <i>t</i> :	4.16*	5.55*
95% C.I. for true difference	1.10 to 3.32	1.42 to 3.15
Intergroup comparison (unpaired <i>t</i>) <i>t</i> = 0.116 (38 df) NS.		

*Significant at $P < 0.001$.
NS = Not significant.

Table 3. Response to Tactile Stimulus (Yeaple probe-cm) (Log₁₀ Transformed Data)

	Silica-Based Group (20)	Diatomaceous Earth Group (20)
Baseline mean (S.E.)	1.142 (0.030)	1.132 (0.035)
20-Week mean (S.E.) (12-week posttreatment)	1.318 (0.053)	1.341 (0.057)
Mean difference (S.E.)	0.176 (0.055)	0.207 (0.055)
Comparison 0 week - 20 week <i>t</i> =	3.452 (19 d.f.)*	3.471 (19 d.f.)*
95% Confidence intervals for true ratio	0.52 to 0.85	0.47 to 0.83
I.e., in the silica group, the tactile response would be expected to be greater at 20 weeks than at baseline by a factor of between 1.18 and 1.92 (1/0.85 and 1/0.52).		
Intergroup comparison of difference <i>t</i> =	0.392 (38 d.f.) NS	
Mean regression coefficient <i>b</i> (S.E.)	0.0175 (0.0042)	0.0218 (0.0047)
Mean level of response (S.E.)	1.26 (0.334)	1.26 (0.041)
<i>t</i> =	0.099 (38 d.f.) NS	

* Significant at $P < 0.01$.
NS = Not significant

regression coefficient for each patient in both groups for the total time period (readings at 0, 2, 4, 8, and 20 weeks). The mean regression coefficients for each group were then compared using an unpaired *t* test (38 degrees of freedom).

Finally, the mean scores of the 5 timed readings at each time point for each patient were computed and the 2 group means compared, again using unpaired *t* tests (38 degrees of freedom) to ensure no bias existed between groups in

terms of the proportion of high or low responses within each group.

RESULTS

No changes were observed in the oral tissues of any subject in either group following cessation of the clinical trial. Twenty-four patients received no dental treatment during the 12 week post-completion period. Of the remaining 16, 9 received scaling and polishing, 4 had teeth restored, 2

Table 4. Subjective Response to Tactile Stimulus Visual Analogue Scale (cm)

	Silica-Based Group (20)	Diatomaceous Earth Group (20)
Baseline mean (S.E.)	3.51 (0.356)	3.46 (0.401)
20-Week mean (S.E.) (12-week posttreatment)	2.35 (0.364)	1.75 (0.342)
Mean difference (S.E.)	1.151 (0.386)	1.707 (0.386)
Comparison 0-20 weeks		
95% Confidence intervals	$t = 3.839$ (19 d.f.)* 0.52 to 1.78	3.744 (19 d.f.)* 0.75 to 2.66
Intergroup comparison of difference		
Mean regression coefficient b (S.E.)	$t = 1.019$ (38 d.f.) NS -0.904 (0.0214)	-0.1069 (0.0311)
Mean level of response (S.E.)	$t = 0.437$ NS 2.55 (0.303)	2.25 (0.259)
	$t = 0.771$ NS	

* Significant at $P < 0.01$.

NS = Not significant.

Table 5. Subjective Response to Cold Stimulus. Dental Air Syringe Visual Analogue Scale (cm)

	Silica-Based Group (20)	Diatomaceous Earth Group (20)
Baseline mean (S.E.)	5.28 (0.284)	5.11 (0.263)
20-Week mean (S.E.) (12-weeks posttreatment)	3.29 (0.452)	3.22 (0.508)
Mean difference (S.E.)	1.992 (0.444)	1.895 (0.444)
Comparison 0-20 wks		
95% Confidence intervals	$t = 4.096$ (19 d.f.)* 0.97 to 3.01	4.764 (19 d.f.)* 1.06 to 2.73
Intergroup comparison of differences		
Mean regression coefficient b (S.E.)	$t = 0.155$ NS -0.1551 (0.0319)	-0.1471 (0.0363)
Mean level of response (S.E.)	$t = 0.166$ NS 3.77 (0.350)	3.70 (0.361)
	$t = 0.139$ NS	

* Significant at $P < 0.01$.

NS = Not significant.

Table 6. Visual Analogue Scale (cm) for Overall Perceived Discomfort to Everyday Stimuli

	Silica-Based Group (20)	Diatomaceous Earth Group (20)
Baseline mean (S.E.)	4.15 (0.429)	4.44 (0.453)
20-Week mean (S.E.) (12-week posttreatment)	2.86 (0.639)	2.43 (0.519)
Mean difference (S.E.)	1.292 (0.545)	2.015 (0.545)
Comparison 0-20 weeks		
95% Confidence intervals	$t = 2.322^*$ 0.13 to 2.45	3.786† 0.90 to 3.13
Intergroup comparison of differences		
Mean regression coefficient b (S.E.)	$t = 0.938$ NS -0.1033 (0.0459)	-0.1600 (0.409)
Mean level of response (S.E.)	$t = 0.921$ NS 2.90 (0.372)	3.09 (0.374)
	$t = 0.350$ NS	

* Significant at $P < 0.05$.† Significant at $P < 0.01$.

NS = Not significant.

had an examination only, and 1 received penicillin for a periodontal abscess. Treatment did not involve any of the study teeth. As patients had not been advised there would be a recall visit at the time of the original study, no reliable information was available concerning subsequent dentifrice use.

The results for the original 8-week clinical study are summarized in Table 2. The results for all variables indicated a remarkably regular trend towards reduction with time but

without any apparent differences between the silica-based (low abrasive) and diatomaceous earth groups. The results for the present study are shown in Tables 3, 4, 5, and 6.

In summary, it was found that for all data there was a significant and favorable change in response in both dentifrice groups over the 12-week post-treatment time period, with neither group showing a significantly higher or lower change compared with the other. The 95% Confidence Intervals (Tables 3, 4, 5, and 6) give the minimum and max-

imum ranges for the mean expected differences likely to be seen in the population from which the samples are drawn. There is less than a 5% chance that the true population mean will lie outside this range. There were no significant differences in the rate of change of response between groups over the 12 week post-treatment period, and no significant differences in the overall level of response.

DISCUSSION

The use of low abrasives in dentifrices formulated for the treatment of CDH has been investigated in several recent clinical trials.^{11,19-21,24} While various in vitro studies have indicated that the use of the low abrasive component silica, in particular, may partly occlude dentinal tubules,^{14,28-30} there appear to be conflicting results from the clinical studies. For example, if reduction in sensitivity can be attributed to the silica-based component,¹¹ then one would expect the group using the precipitated silica dentifrice to have demonstrated a significant difference in reduction of sensitivity. The results from the original 8-week study, however, demonstrated that the 2 SCH dentifrices were equally effective in reducing CDH (Tables 2, 3, 4, 5, and 6), and confirm McFall and Hamrick's conclusions²⁰ with respect to the role of the abrasive components. Although the role of the abrasive component cannot be ruled out, the conflicting results from the various clinical studies highlight discrepancies between laboratory and clinical findings which may not necessarily favor the former.

Few studies have published data following cessation of dentifrice use. Addy et al.¹¹ reported a reversal in sensitivity levels towards baseline following cessation. In the original 8-week study for both tactile sensitivity to probe and tactile VAS scores, the results indicated a remarkably regular trend towards reduction with time, but without any apparent differences between the silica-based (low abrasive) and diatomaceous earth groups at any time point, whereas results from the 20-week study appear to show a very slight trend towards an increase in sensitivity with time, again without any apparent or detectable differences between the 2 groups (Table 3 and 4), but with reductions in sensitivity compared to baseline still evident at 20 weeks. As with mean probe and tactile VAS scores, both air mean VAS scores and overall sensitivity VAS scores showed a regular trend towards reduction in sensitivity to cold and everyday stimuli respectively, with time, during the 8-week controlled study, without any apparent differences between groups (Table 2); whereas results from the 20-week study appear to show slight regression following cessation of dentifrice use, again without any apparent or detectable differences between the groups (Tables 5 and 6). Reductions in sensitivity to cold as well as reductions in overall sensitivity compared to baseline, however, were still evident at 20 weeks.

On the basis of the 3 accepted methods of assessment, the results overall would indicate that there was a very slight and not statistically significant change in mean sensitivity

levels following the end of the original clinical study. Although there was a very slight reversal of the trend demonstrated during the original study, no apparent differences were detected between the silica-based low abrasive and diatomaceous earth groups at the 20-week time point, and overall, sensitivity remained significantly lower than at baseline.

It was concluded that reductions in sensitivity achieved by the use of both dentifrices were still evident 3 months after the cessation of their regular controlled use, and that the abrasivity of the dentifrice did not affect its desensitizing activity.

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REFERENCES

1. Blitzer, B. A consideration of the possible causes of dental hypersensitivity. Treatment by a strontium-ion dentifrice. *Periodontics* 1967;5:318-321.
2. Shapiro WB, Kaslick RS, Chasens AI, Weinstein D. Controlled clinical comparison between a strontium chloride and a sodium monofluorophosphate toothpaste in diminishing root hypersensitivity. *J Periodontol* 1970;41:523-525.
3. Shapiro WB, Kaslick RS, Chasens AI. The effect of a strontium chloride toothpaste on root sensitivity in a controlled clinical study. *J Periodontol* 1970;41:702-703.
4. Carrasco PH. Strontium chloride toothpaste. Effectiveness as related to duration of use. *Pharmacol Ther Dent* 1971;1:209-215.
5. Hernandez F, Mohammed CO, Shannon I, Volpe A, King W. Clinical study evaluating the desensitizing effect and duration of two commercially available dentifrices. *J Periodontol* 1972;43:367-372.
6. Uchida A, Wakano Y, Fukuyama O, Miki T, Iwayama Y, Okada H. Controlled clinical evaluation of 10% strontium chloride dentifrice in treatment of dentin hypersensitivity following periodontal surgery. *J Periodontol* 1980;51:578-581.
7. Collins JF, Gingold J, Stanley H, Simring M. Reducing dentinal hypersensitivity with strontium chloride and potassium nitrate. *Gen Dent* 1984;32:40-43.
8. Council on Dental Therapeutics. Acceptance of Sensodyne toothpaste for sensitive teeth. *J Am Dent Assoc* 1985;110:394.
9. Minkoff S, Axelrod S. Efficacy of strontium chloride in dental hypersensitivity. *J Periodontol* 1987;58:470-474.
10. Clark DC, Hanley SP, Montey JA, Geoghegan S, Vinet D. The effectiveness of a fluoride varnish and a desensitizing toothpaste in treating dentine sensitivity. Effects of sodium citrate and sodium ingredients. *J Periodont Res* 1985;20:89-93.
11. Addy M, Mostafa P, Newcombe R. Dentine hypersensitivity. A comparison of five toothpastes used during a 6-week treatment period. *Brit Dent J* 1987;163:45-51.
12. Greenhill JD, Pashley DH. The effects of desensitizing agents on the hydraulic conductance of human dentin *in vitro*. *J Dent Res* 1981;60:686-698.
13. Mostafa P, Addy M, Morgan T. Scanning electron microscopic, x-ray diffraction analysis, atomic absorption and fluoride probe measurements of the uptake of toothpaste ingredients onto dentine. *J Dent Res* 1983;62(Spec. Issue):433 (Abstr. 165).
14. Pashley DH. Smear layer; physiological considerations. *Oper Dent* 1984;3(Suppl.):13-29.
15. Pashley DH. Dentin permeability, dentin sensitivity and treatment through tubular occlusion. *J Endodont* 1986;12:465-474.

16. Pashley DH, O'Meara JA, Kepler EE, Galloway SE, Thompson SM, Stewart EP. Dentin permeability. Effects of desensitizing dentifrices *in vitro*. *J Periodontol* 1984;55:522-525.
 17. Pashley DH, Leibach JG, Horner JA. The effects of burnishing NaF/Kaolin/glycerin paste on dentin permeability. *J Periodontol* 1987;58:19-23.
 18. Addy M, Loyn T, Adams D. The effects *in vitro* of some mouthwash products on the dentine smear layer. *J Dent Res* 1990;69(Spec. Issue):699(Abstr. 89).
 19. Manoechehr-Pour M, Bhat M, Bissada N. Clinical evaluation of two potassium nitrate toothpastes for the treatment of dental hypersensitivity. *Periodont Case Reports* 1984;6:25-30.
 20. McFall WT, Hamrick SW. Clinical effectiveness of a dentifrice containing fluoride and a citrate buffer system for treatment of dentinal hypersensitivity. *J Periodontol* 1987;58:701-705.
 21. Silverman G. The sensitivity-reducing effect of brushing with a potassium nitrate-sodium monofluorophosphate dentifrice. *Comp Cont Educ Dent* 1985;6:131-133.
 22. Salvato A, Clark G, Curro FA. Clinical efficacy of Sensodyne-F as a desensitizing dentifrice. *J Dent Res* 1989;68(Spec. Issue): 971(Abstr. 839).
 23. Jackson RJ, Duke SA, Wicks MA. The evaluation of two dentifrices formulated for the treatment of dentine hypersensitivity. *J Dent Res* 1989;68(Spec. Issue):602(Abstr. 346).
 24. Jackson RJ, Duke SA, Wicks MA, McDonald FE. The effects of antisensitivity dentifrices on subjects consuming acid diets. *J Dent Res* 1990;69(Spec. Issue):988(Abstr. 268).
 25. Gillam DG, Newman HN, Davies EH, Bulman JS. Clinical efficacy of a low abrasive dentifrice for the relief of cervical dentinal hypersensitivity. *J Clin Periodontol* (In Press).
 26. Loe H, Silness J. Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odontol Scand* 1963;21:533-551.
 27. Polson AM, Caton JG, Yeaple RN, Lander HA. Histological determination of probe tip penetration into gingival sulcus of humans using an electronic pressure sensitive probe. *J Clin Periodontol* 1980;7:479-488.
 28. Mostafa P. University of Wales College of Medicine. Dentine hypersensitivity: Clinical and laboratory investigations. [Dissertation] 1985;164-167.
 29. Mostafa P, McCarthy D, Addy M. A comparison of 3 test silica based toothpastes and 2 commercially desensitizing toothpastes on dentine hypersensitivity (DH). *J Dent Res* 1986;65(Spec. Issue): 497(Abstr. 90).
 30. Absi EG, Addy M, Adams D. Dentine hypersensitivity. Uptake of various desensitizing toothpastes on dentine *in vitro* S.E.M. investigation. *J Dent Res* 1989;68(Spec. Issue): 573(Abstr. 117).
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Clinical efficacy of a low abrasive dentifrice for the relief of cervical dentinal hypersensitivity

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Abstract. 2 strontium chloride hexahydrate-containing dentifrices (SCH), similar except for their respective abrasive systems, were compared in a 2-month randomised double-blind parallel clinical study to evaluate their comparative effectiveness in terms of cervical dentinal hypersensitivity. 2 groups of 20 subjects, each with cervical dentinal hypersensitivity, were evaluated for tactile sensitivity by Yeaple probe, air sensitivity using a dental air syringe and subjective perception of pain by means of a visual analogue scale. There was no difference between the dentifrices as regards reduction of cervical dentinal hypersensitivity at each time point. The response to both dentifrices was evident within 4 weeks of use and the degree of improvement increased throughout the 8-week study period. The results support the conclusion that changing the abrasive component of SCH dentifrices did not significantly increase or decrease the (desensitizing) activity of the original product.

Key words: dentinal hypersensitivity; strontium chloride; abrasive; pain.

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Strontium chloride hexahydrate (SCH) has been widely used in a dentifrice form for the treatment of cervical dentinal hypersensitivity. SCH appears to act both as a protein precipitant and a tubule occluding agent (Cohen 1961, Skurnik 1963, Blitzer 1967, Gedalia et al. 1978, Uchida et al. 1980). Several workers have shown that SCH causes deposition of an insoluble barrier at cervical dentinal tubule orifices (Ross 1961, Blitzer 1967, Gedalia et al. 1978), whilst Kun (1976) demonstrated in vitro that SCH produced significant penetration of tubules. Further in vitro studies (Greehill & Pashley 1981, Mostafa et al. 1983, Pashley et al. 1984, Addy et al. 1990) however, suggested that these results were attributable, not to the active ingredient per se, but rather to the abrasive components of the dentifrice, notably silica, contributing to the formation of a smear layer which effectively blocks the exposed tubule orifices (Pashley 1984, 1986, Pashley et al. 1987).

Others (Clark et al. 1985, Addy et al. 1987) questioned the effectiveness of SCH in reducing cervical dentinal hypersensitivity (CDH). Recent studies (Addy et al. 1987, Jackson et al. 1989, 1990) comparing a silica-based product containing strontium acetate and fluo-

ride (SrAc₂F) with Sensodyne® containing SCH and the abrasive diatomaceous earth reported that the SrAc₂F dentifrice was more effective in controlling CDH than the SCH product. The purpose of this present clinical study, therefore, was to compare the efficacy of two anti-sensitivity strontium chloride hexahydrate-based dentifrices differing only in their respective abrasive systems.

Material and Methods

49 patients were originally enrolled into the study. Nine were subsequently excluded. One failed to disclose a medical problem, one had periodontitis, one did not respond to the test stimuli, one failed to return following screening and five were unable to attend for all visits. Forty subjects completed the eight week clinical study. The investigation was a double-blind, 2-way comparative parallel study of 40 patients with a mean age of 42.8 years. Subjects were randomly assigned to one of two treatment groups using a computer-generated randomisation code.

Inclusion criteria

Selection of subjects was restricted to individuals who presented with a hyper-

sensitivity complaint, accompanied by cervical erosion, abrasion and/or gingival recession on at least one tooth for tactile stimulus and two for cold air stimulus on suitable teeth anterior to the second molar. Sensitive teeth without restorations were preferred, although teeth with restorations were included provided the restorations were no greater than one half of the distance through dentine in anterior, premolar and first molar teeth. Any restoration margins were at least 5 mm from the area of sensitivity. Decision to include such teeth was made on the basis of clinical as well as radiographical (OPT) evaluation. Subjects were included who, during the baseline examination, experienced sensitivity to a tactile force of 10–50 g and recorded a Visual Analogue Scale score of 3–8 cm following application of a cold air stimulus. All showed at least one tooth sensitive to the Yeaple probe and two to the dental air syringe, although not necessarily the same teeth.

Exclusion criteria

Subjects with chronic systemic disease or a history of gingival surgery within the previous 6 months were excluded, as were patients who were pregnant or

lactating, or who were on any medication. Teeth with suspected pulpitis, caries or cracked enamel were excluded, as were all teeth with defective restorations and those used as denture abutments. Subjects with gingival index (Löe & Silness 1963) ≥ 1 for the gingivae of prospective sensitive and adjacent teeth, or ≥ 2 for non-study teeth were also excluded. Subjects using a desensitising dentifrice agreed to refrain from using same for at least 2 months prior to the trial. 14 subjects (7 male, 7 female) were in this category and a fluoride dentifrice was substituted for the desensitising paste. The subjects continued their normal daily oral hygiene.

Screening

Following approval of the Institute and Hospital Joint Research and Ethics Committee and individual voluntary written informed consent, subjects completed a questionnaire concerning their hypersensitivity condition. This was confirmed clinically by the investigator (DGG). Subjects thus screened were examined for baseline sensitivity using both tactile and cold air stimuli. Sensitive teeth were initially detected with a no. 6 straight probe cervically on each tooth anterior to the second molar. 10 minutes later, the investigator assessed the tooth response to cold air using the standard dental air syringe at 40–65 psi at a temperature of 19°C.

Procedure for measuring hypersensitivity

Tactile method (Yeaple Probe – modified)

The Yeaple probe (Vine Valley Research, Middlesex, NY, USA) was modified to accept a tip with rounded end 0.30 mm–0.35 mm diameter (Williams 14W). The probe is designed to deliver a pre-set force when the tip is applied perpendicular to the cervical labial surface (Polson et al. 1980). Application of the incremental probe force was continued to the point at which discomfort was just felt and the force setting noted. The maximum force applied was 70 g. If, following the 2 baseline measurements, the subject did not perceive any discomfort at that force, a score of 70 was recorded. The subject was also asked to rate the perception of sensitivity experienced during applications of tactile probe by placing a mark on a 10 cm line on a "tactile sensi-

tivity scale form". The distance of the mark from the "no pain" end provided an estimate of pain perceived by the subject and constituted a tactile VAS score. The Yeaple probe was calibrated prior to each clinical session using a Sartorius 1202 MP top loading digital balance (Brinkman Instruments Co., Division of Sybron, Westbury, NY, USA) to obtain a correlation of the probe meter readings in DC micro amperes and the grams of force.

Cold air (thermal method)

10 min later, response was assessed to a 1 s application from a standard dental unit syringe at 40–65 psi at a temperature of 19°C, and directed perpendicular to the exposed root surface after isolating the test tooth. Using the principle of visual analogue scale scores (0–10) air pain intensity was indicated by the subject placing a mark on a 10 cm line on a "subject air sensitivity score form". The distance of the mark from the "no pain" end provided an estimate of pain perceived by the subject and constituted an air sensitivity score.

Subjective reporting of pain-baseline

Subjects were asked to rate their perception of sensitivity to hot/cold food and drink, air, toothbrushing and sweet and sour food by placing a mark on a 10 cm line. The distance of the mark from the "no pain" end provided an estimate of the overall severity of pain perceived by the subject. After approximately one week, a second baseline determination was made repeating the above procedure.

Test product assignment

Assignment of subjects to experimental cells was by a computer-generated random number code. Each individual coded kit contained two toothbrushes (Sensodyne Search 4) and tubes (3 × 45 ml) of one of the test dentifrices. Dentifrices were closely matched with respect to taste, colour, consistency and appearance and dispensed double-blind. Subjects were directed to brush twice each day, morning and evening, in their usual manner, with the brush supplied, for 56 consecutive days, using only the assigned dentifrice.

Each subject was instructed to place an inch length of toothpaste on the wet toothbrush and to brush all surfaces of all teeth for at least 1 min before ex-

pectorating. Each subject recorded his/her daily brushing in a diary which was provided. All assigned products were weighed before and after use by the investigator to assist in determining compliance. The diaries were checked at each visit by a 3rd party who also distributed the assigned products. All patients attended all appointments, and on or close to day 56 with residual toothpaste. Recorded non-compliance with regard to dentifrice use was rare..

Data analysis

All data were tested for normality using a normal scores transformation and plotting the result against the original data. A normal distribution was indicated by a reasonably straight line plot with no marked concavity or convexity. All data proved to be normally distributed with the exception of tactile force which was skewed to the right. A logarithmic transformation was, therefore, used to normalise these data and stabilise the variance. For this variable, therefore, descriptive statistics only were provided for the raw (untransformed) data and analysis was carried out on the log-transformed data. Normality tests also detected a marked "outlier" reading in the test group "baseline minus 2-week" data. Analyses were, therefore, performed both excluding and including the "outlier". In the event, inclusion did not affect the overall trend of the data, but data excluding the "outlier" were taken as being more reliable.

Subject-based (n: no. of subjects)

Paired *t* tests were utilised for each treatment cell to determine if differences between readings at baseline and at scheduled examination times were statistically significant at the 95% confidence level. Similarly, at each time point any differences between the dentifrices and their effects on sensitivity scores were tested for statistical significance by means of a two-sample *t* test. Confidence intervals were also calculated and only probabilities of less than or equal to 0.05 were considered to indicate a significant difference between means.

Results

40 subjects (15 male and 25 female, mean age 42.8 ± 8.2 years, completed this study (Table 1). No changes were seen in the oral tissues of any subject

Table 1. Patient data

	Test group		Control group		Total	
	N	age ($\bar{x} \pm \text{SD}$) (years)	N	age ($\bar{x} \pm \text{SD}$) (years)	N	age ($\bar{x} \pm \text{SD}$) (years)
female	13	42.6 \pm 11.38	12	43.9 \pm 8.24	25	43.2 \pm 9.81
male	7	40.3 \pm 4.88	8	43.8 \pm 3.84	15	42.1 \pm 4.57
mean	20	41.8 \pm 9.52	20	43.8 \pm 6.69	40	42.8 \pm 8.18

Test = silica-based. Control = diatomaceous earth. N = number of patients. \bar{x} = mean. SD = standard deviation.

Table 2. Response to tactile stimulus (Yeaple probe-gm) (\log_{10} transformed data)

	Test group (n = 20)		Control group (n = 20)	
	$\bar{x} \pm \text{SD}$	% change	$\bar{x} \pm \text{SD}$	% change
baseline	1.14 \pm 0.133	0	1.13 \pm 0.157	0
2 weeks	1.19 \pm 0.153	-3.9	1.14 \pm 0.169	-0.7
4 weeks	1.26 \pm 0.200	-10.6	1.25 \pm 0.285	-10.5
8 weeks	1.40 \pm 0.257	-22.5	1.41 \pm 0.275	-24.6

Test = silica-based. Control = diatomaceous earth.

in either group over the 8-week study period, nor were side-effects or untoward reactions reported to or observed by the investigator.

Probe evaluation

Baseline scores for both tactile sensitivity to the Yeaple probe (Table 2) and tactile VAS (Table 3) were compared for the two groups and found to exhibit no significant differences. Mean probe scores for the silica-based group (test) (\log -transformed) increased in relation to baseline, indicating a decrease in sensitivity. The 2-week increase was not significant (95% CI for the ratio: 0.77 to 1.05), while those for the 4-week and 8-week increases were significant (95% CI for the ratio: 0.60 to 0.95 and 0.42 to 0.74, respectively). For the control group, the mean probe scores increased. The 2-week increment was again not significant (95% CI for the ratio: 0.85 to 1.12), but the 4-week and 8-week increments were significant (95% CI: 0.60 to 0.96, and 0.41 to 0.67). Mean tactile VAS scores (excluding outlier) for the silica-based group (test) decreased in re-

lation to baseline. The 2-week and 8-week decreases were very highly significant (95% CI for the difference between the means: 0.55 to 1.53 and 0.83 to 2.42) while the 4-week decrease was highly significant (95% CI: 0.44 to 1.92). For the control group mean tactile VAS scores, the 2-week and 8-week decreases were again very highly significant (95% CI for the difference between the means: 0.65 to 1.78, and 0.92 to 2.44) while the 4-week decrease was highly significant (95% CI: 0.40 to 2.55). For both tactile sensitivity to probe and tactile VAS scores, the results indicated a regular trend towards reduction in sensitivity with time, but without any apparent or detectable differences between the groups (Tables 2, 3).

Cold air sensitivity

As with tactile sensitivity, air sensitivity values were indistinguishable between the groups at baseline (Table 4). Mean VAS scores for test and control groups decreased, all decrements differing significantly from 0 (95% CI: 0.24 to 2.44, 0.59 to 2.78, and 1.41 to 3.70 (test

group), (95% CI: 0.45 to 1.63, 1.08 to 2.65 and 1.32 to 3.21 control group). There were no inter-group significant differences at any time-interval. These results again indicated a regular trend towards reduction in sensitivity to cold with time, but without any apparent or detectable differences between the groups (Table 4).

Subjective evaluation: overall sensitivity VAS

Overall sensitivity VAS score values were indistinguishable between the groups at baseline (Table 5). Mean VAS decrements differed significantly from 0 (95% CI: 0.045 to 2.05, 0.64 to 2.74 and 1.10 to 3.32 test), (95% CI: 0.34 to 1.94, 0.22 to 2.46 and 1.42 to 3.15 control). There were no inter-group significant differences at any time interval. As with the other variables there was a regular trend towards reduction in sensitivity with time, but without any apparent difference between the groups (Table 5).

Discussion

The 2 SCH dentifrices used in the present study were similar except for their respective abrasive systems. One contained diatomaceous earth and the other precipitated silica. If the reduction in sensitivity was attributable to the silica-based component (Addy et al. 1987), then one would have expected the group using the precipitated silica dentifrice to have demonstrated a significant difference in reduction of sensitivity. The results of the present study, however, demonstrate that the two SCH dentifrices were equally effective (Tables 2-5), and confirm McFall & Hamrick's (1987) conclusions with respect to the role of the abrasive components. These results also highlight the discrepancy that may be observed between laboratory and clinical findings, which may not necessarily favour the former.

Problems in evaluating the effectiveness of a dentifrice in a clinical trial may derive from a lack of predictable, reliable and reproducible methodology for evaluating the subjective response of the patient, which can be further modified by social, psychological and situational factors (McGrath 1986). Hence there are a variety of methods used to evaluate CDH, e.g. mechanical and thermal stimuli and patient's subjective assessment of pain in response to normal daily stimuli (Green et al. 1977,

Table 3. Subjective response to tactile stimuli: visual analogue scale scores (cm)

	Test group (n = 20)		Control group (n = 20)	
	$\bar{x} \pm \text{SD}$	% change	$\bar{x} \pm \text{SD}$	% change
baseline	3.5 \pm 1.59	0	3.5 \pm 1.79	0
2 weeks*	2.6 \pm 1.36	-25.6	2.3 \pm 1.38	-35.1
4 weeks	2.3 \pm 1.73	-33.6	2.0 \pm 1.75	-42.5
8 weeks	1.9 \pm 1.57	-46.3	1.8 \pm 1.27	-48.5

*One outlier eliminated from 2-week data for test group.

Table 4. Subjective response to cold stimuli (dental air syringe): visual analogue scale scores (cm)

	Test group (n=20) $\bar{x} \pm SD$	% change	Control group (n=20) $\bar{x} \pm SD$	% change
baseline	5.3 \pm 1.27	0	5.1 \pm 1.18	0
2 weeks	3.9 \pm 1.97	-25.4	4.1 \pm 1.91	-20.4
4 weeks	3.6 \pm 2.26	-31.9	3.2 \pm 1.79	-36.6
8 weeks	2.7 \pm 2.27	-48.3	2.9 \pm 2.56	-44.3

Table 5. Visual analogue scale scores (cm) for overall perceived discomfort to everyday stimuli

	Test group (n=20) $\bar{x} \pm SD$	% change	Control group (n=20) $\bar{x} \pm SD$	% change
baseline	4.2 \pm 1.92	0	4.4 \pm 2.03	0
2 weeks	3.1 \pm 2.30	-25.2	3.3 \pm 2.09	-26.7
4 weeks	2.5 \pm 1.94	-40.7	3.1 \pm 2.14	-30.2
8 weeks	1.9 \pm 1.93	-53.2	2.2 \pm 1.96	-51.4

Tarbet et al. 1979, 1980, 1982, Minkoff et al. 1975, Uchida et al. 1980). Opinions vary as to the reliability of the various methods of assessment (Green et al. 1977, Addy & Dowell 1983, Lecointre et al. 1986, Addy et al. 1987). More recently efforts have been made to develop controlled reproducible stimuli more suited to the evaluation of CDH, for example, the Yeaple probe, the Yeh and Temptronic devices, and thermal probes (Silverman 1985, Minkoff & Axelrod 1987, Addy et al. 1987, Clarke et al. 1987, Ong & Strahan 1989, Person et al. 1989). In the present study, the Yeaple probe (quantifiable) and cold air stimuli (semi-quantifiable) were utilised, together with a subject assessment of pain using VAS scores. All appeared satisfactory for the measurement of subject response (Tables 2–5).

Positive placebo effects have also been reported in clinical trials (Addy & Dowell 1983) and participants using control pastes with no active ingredients have experienced significant reductions in their perception of sensitivity. The placebo effect, together with recognised associated non-placebo effects, e.g., an improvement in the participant's oral hygiene (Peden 1977) and possible natural desensitization with time (Karlson & Penney 1975), may also contribute to a reduction in sensitivity.

While a placebo effect was possible in the present study, the study was randomised and double-blind, and patients were in no way informed in a manner which would have implied efficacy for either dentifrice. Further, SCH dentifrice produced significantly greater re-

duction in sensitivity than a placebo, using a similar range of assessment methods (Minkoff & Axelrod 1987). In any event, it is doubtful practice to refuse treatment for patients suffering pain, any more than would be the case for a fluoride dentifrice in a caries study.

The results for this randomised double-blind parallel study of 40 patients with cervical dentinal hypersensitivity over 8 weeks of product use demonstrated that when assessed by tactile and cold air stimuli, together with patients' subjective response, the SCH dentifrices were equally effective and seemed to act to the same degree in relation to time. The response to both dentifrices was evident within 4 weeks of use and the degree of improvement increased during the duration of the 8-week study. In conclusion, the results suggest that changing the abrasive component of SCH dentifrices did not significantly increase or decrease the desensitizing activity of the original product.

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Zusammenfassung

Klinische Wirksamkeit einer Zahnpaste mit geringen abrasiven Eigenschaften bei der Linderung zervikaler Dentinüberempfindlichkeit Bei 2 Strontiumchlorid enthaltenden Zahn-

pasten (SCH), die, abgesehen von unterschiedlichen abrasiven Eigenschaften, einander ähnlich waren, wurde ihre Wirksamkeit bei der Linderung zervikaler Dentinhyper-sensibilität mit Hilfe einer randomisierten, 2 Monate langen klinischen Doppelblindunter-suchung verglichen. Bei 2 Gruppen von je 20 Probanden mit zervikaler Dentinüber-empfindlichkeit wurde die taktile Sensitivität mit einer Yeaple Sonde (Methode zur taktilen Hypersensitivitätsmessung; siehe im Text) be-urteilt. Ausserdem wurde die Empfindlichkeit gegenüber einem Luftstrom mit dem zahn-ärztlichen Luftbläser getestet und die subjektive Schmerzempfindung mit einer visuellen Analogskala registriert. An keinem Zeit-punkt der Messungen lagen zwischen den bei-den Zahnpasten Unterschiede hinsichtlich ihrer Reduktion der zervikalen Dentinüber-empfindlichkeit vor. Bereits nach 4 Wochen war bei beiden Zahnpasten eine desensibili-sierende Wirkung deutlich merkbar, die sich während der 8-wöchentlichen Untersuchungsperiode stetig verstärkte. Die Resultate lassen die Schlußfolgerung zu, daß eine Ver-änderung der abrasiven Komponente bei SCH-Zahnpasten die desensibilisierende Eigenschaft des Originalproduktes weder sig-nifikant erhöht noch senkt.

Résumé

Efficacité d'un dentifrice peu abrasif à soulager l'hypersensibilité dentinaire cervicale Deux dentifrices contenant de l'hexahydrate chlorure de strontium (SCH) et ne différant que par leur degré d'abrasion ont été comparés lors d'une étude randomisée à double insu ayant duré 2 mois. Deux groupes de 20 sujets avec hypersensibilité dentinaire cervicale ont été évalués pour leur sensibilité tactile à l'aide de la sonde Yeaple, leur sensibilité à l'air avec une seringue dentaire à air et leur perception subjective de la douleur grâce à une échelle analogue visuelle. Aucune différence entre les deux dentifrices n'a été mise en évidence. La réponse aux deux dentifrices était manifeste après 4 semaines d'utilisation et le degré d'amélioration augmentait pendant les 8 semaines de l'étude. Changer le degré d'abrasion des dentifrices SCH ne fait pas varier significativement le soulagement de l'hypersensibilité dentinaire cervicale apporté par le produit initial.

References

- Addy, M. & Dowell, P. (1983) Dentine hyper-sensitivity – A review. Clinical and in vitro evaluation of treatment agents. *Journal of Clinical Periodontology* **10**, 351–363.
- Addy, M., Loyn, T. & Adams, D. (1990) The effects in vitro of some mouthwash products on the dentine smear layer. *Journal of Dental Research* **69**, 89 (abstr.).
- Addy, M., Mostafa, P. & Newcombe, R. (1987) Dentine hypersensitivity. A comparison of five toothpastes used during a 6-week treatment period. *British Dental Journal* **163**, 45–51.

- Blitzer, B. (1967) A consideration of the possible causes of dental hypersensitivity. Treatment by a strontium-ion dentifrice. *Periodontics* **5**, 318–321.
- Clark, D. C., Hanley, J. A., Geoghegan, S. & Vinet, D. (1985) The effectiveness of a fluoride varnish and a desensitizing toothpaste in treating dentinal hypersensitivity. *Journal of Periodontal Research* **20**, 212–219.
- Clark, D. C., Al-Joburi, W. & Chan, E. C. S. (1987) The efficacy of a new dentifrice in treating dentin sensitivity. Effects of sodium citrate and sodium ingredients. *Journal of Periodontal Research* **22**, 89–93.
- Cohen, A. (1962) Preliminary study of the effects of a strontium chloride dentifrice for the control of hypersensitive teeth. *Oral Surgery, Oral Medicine, Oral Pathology* **14**, 1046–1052.
- Gedalia, I., Brayer, L., Kalter, N., Richter, M. & Stabholz, A. (1978) The effect of fluoride and strontium application on dentin: in vivo and in vitro studies. *Journal of Periodontology* **49**, 269–272.
- Green, B. L., Green, M. L. & McFall, W. T. (1977) Calcium hydroxide and potassium nitrate as desensitizing agents for hypersensitive root surfaces. *Journal of Periodontology* **48**, 667–672.
- Greenhill, J. D. & Pashley, D. H. (1981) The effects of desensitizing agents on the hydraulic conductance of human dentin in vitro. *Journal of Dental Research* **60**, 686–698.
- Jackson, R. J., Duke, S. A. & Wicks, M. A. (1989) The evaluation of two dentifrices formulated for the treatment of dentine hypersensitivity. *Journal of Dental Research* **68**, 603 (abstr.).
- Jackson, R. J., Duke, S. A., Wicks, M. A. & McDonald, F. E. (1990) The effects of anti-sensitivity dentifrices on subjects consuming acid diets. *Journal of Dental Research* **69**, 368 (abstr.).
- Karlsson, U. L. & Penney, D. A. (1975) Natural desensitization of exposed tooth roots in dogs. *Journal of Dental Research* **54**, 982–986.
- Kun, L. (1976) Etude biophysique des modifications des tissus dentaires provoquées par l'application locale de strontium. *Revue Mensuelle Suisse d'Onto-Stomatologie* **86**, 661–676.
- Lecointre, C., Apiou, J., Marty, P. & Poitou, P. (1986) Controlled trial of the action of a toothpaste containing nicomethanol hydrofluoride in the treatment of dentine hypersensitivity. *Journal of Internal Medicine Research* **14**, 217–222.
- Löe, H. & Silness, J. (1963) Periodontal disease in pregnancy. 1. Prevalence and severity. *Acta Odontologica Scandinavica* **21**, 533–551.
- McFall, W. T. & Hamrick, S. W. (1987) Clinical effectiveness of a dentifrice containing fluoride and a citrate buffer system for treatment of dentinal sensitivity. *Journal of Periodontology* **58**, 701–705.
- McGrath, P. A. (1986) The measurement of human pain. *Endodontics and Dental Traumatology* **2**, 124–129.
- Minkoff, S. & Axelrod, S. (1987) Efficacy of strontium chloride in dental hypersensitivity. *Journal of Periodontology* **58**, 470–474.
- Minkov, B., Marmari, I., Gedalia, I. & Garfunkel, A. (1975) The effectiveness of sodium fluoride treatment with or without iontophoresis on the reduction of hypersensitive dentin. *Journal of Periodontology* **46**, 246–249.
- Mostafa, P., Addy, M. & Morgan, T. (1983) Scanning electronmicroscopic, X-ray diffraction analysis, atomic absorption and fluoride probe measurements of the uptake of toothpaste ingredients onto dentine. *Journal of Dental Research* **62**, 433 (abstr.).
- Ong, G. & Strahan, J. D. (1989) Effects of a desensitizing dentifrice on dentinal hypersensitivity. *Endodontics and Dental Traumatology* **5**, 213–218.
- Pashley, D. H. (1984) Smear layer: physiological considerations. *Operative Dentistry Supplement* **3**, 13–29.
- Pashley, D. H. (1986) Dentin permeability, dentin sensitivity, and treatment through tubule occlusion. *Journal of Endodontics* **12**, 465–474.
- Pashley, D. H., O'Meara, J. A., Kepler, E. E., Galloway, S. E., Thompson, S. M. & Stewart, F. P. (1984) Dentin permeability. Effects of desensitizing dentifrices in vitro. *Journal of Periodontology* **55**, 522–525.
- Pashley, D. H., Leibach, J. G. & Horner, J. A. (1987) The effects of burnishing NaF/kaolin/glycerin paste on dentin permeability. *Journal of Periodontology* **58**, 19–23.
- Peden, J. W. (1977) Dental hypersensitivity. The Journal of the Western Society of Periodontology. *Periodontal Abstracts* **25**, 75–83.
- Person, P., Demand, E. E., Koltun, L. & Spindel, L. M. (1989) A micro-processor temperature-controlled air delivery system for dentinal hypersensitivity testing. *Clinical Preventive Dentistry* **11**, 3–9.
- Polson, A. M., Caton, J. G., Yeaple, R. N. & Lander, H. A. (1980) Histological determination of probe tip penetration into gingival sulcus of humans using an electronic pressure sensitive probe. *Journal of Clinical Periodontology* **7**, 479–488.
- Ross, M. R. (1961) Hypersensitive teeth. Effect of strontium chloride in a compatible dentifrice. *Journal of Periodontology* **32**, 49–53.
- Silverman, G. (1985) The sensitivity-reducing effect of brushing with a potassium nitrate-sodium monofluorophosphate dentifrice. *Compendium of Continuing Education in Dentistry* **6**, 131–136.
- Skurnik, H. (1963) Control of dental hypersensitivity. Preliminary report on a strontium-containing dentifrice. *Journal of Periodontology* **34**, 183–185.
- Tarbet, W. J., Silverman, G., Stolman, J. M. & Fratarcangelo, P. A. (1979) An evaluation of two methods for the quantitation of dentinal hypersensitivity. *Journal of the American Dental Association* **98**, 914–918.
- Tarbet, W. J., Silverman, G., Stolman, J. M. & Fratarcangelo, P. A. (1980) Clinical evaluation of a new treatment for dentinal hypersensitivity. *Journal of Periodontology* **51**, 535–540.
- Tarbet, W. J., Silverman, G., Fratarcangelo, P. A. & Kanapka, J. A. (1982) Home treatment for dentinal hypersensitivity. A comparative study. *Journal of the American Dental Association* **105**, 227–330.
- Uchida, A., Wakano, Y., Fukuyama, O., Miki, T., Iwayama, Y. & Okada, H. (1980) Controlled clinical evaluation of a 10% strontium chloride dentifrice in treatment of dentin hypersensitivity following periodontal surgery. *Journal of Periodontology* **51**, 578–581.

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The effect of strontium chloride hexahydrate dentifrices on plaque accumulation and gingival inflammation

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Abstract. 2 strontium chloride hexahydrate-containing dentifrices (SCH), similar except for their respective abrasive systems, diatomaceous earth or silica-based, were compared for their effects on plaque accumulation and gingival inflammation as part of a 2-month randomised double-blind parallel clinical study. No attempt was made to change the patients' oral hygiene prior to participation in the study. Plaque was assessed using the Silness & Loe index and the gingival condition by the Loe & Silness index GI. There was a slight and non-significant increase in plaque accumulation at 2 weeks from baseline, but relatively negligible change thereafter, the effect being identical in both groups. Similarly, the gingival condition showed a slight index change from baseline, although it tended to be slightly higher in the diatomaceous earth group. The results do not support the conclusions of previous studies which indicated that SCH dentifrices increased plaque accumulation. Neither plaque accumulation nor gingival condition significantly changed from baseline levels during the course of the study.

Key words: dentinal hypersensitivity; strontium chloride; abrasive; plaque; oral hygiene; gingivitis.

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Strontium chloride hexahydrate (SCH) dentifrices with the abrasive diatomaceous earth have been widely used for the treatment of cervical dentinal hypersensitivity (CDH) (Blitzer 1967, Shapiro et al. 1970a, b, Carrasco-P 1971, Hernandez et al. 1972, Uchida et al. 1980, Collins et al. 1984, Minkoff & Axelrod 1987). There have been fewer studies of their effects on plaque. Several investigators have claimed that silica-based products containing strontium acetate and fluoride ($\text{Sr Ac}_2\text{F}$) were more effective in reducing plaque than a SCH dentifrice containing the abrasive diatomaceous earth (Jackson et al. 1989, Addy et al. 1990). The purpose of the present study was therefore to evaluate whether levels of plaque and gingival inflammation were affected by 2 antisensitivity dentifrices differing only in their abrasivity.

Material and Methods

40 subjects, 15 male and 25 female, mean age 42.8 ± 8.2 years participated in the study (Table 1), the details of

which have been described previously (Gillam et al. 1991). Basically, the study groups comprised 40 patients who were assigned to the respective test or control group using a computer-generated random number code. Each patient received a coded kit which contained 2 toothbrushes (Sensodyne Search 4) and tubes (3×45 ml) of 1 of the assigned dentifrices at baseline 2 and 4 weeks.

Dentifrices were closely matched with respect to taste, colour and consistency, and were dispensed in a double-blind manner. Subjects were directed to brush $2 \times$ each day, morning and evening, in their usual manner with the brush supplied for 56 consecutive days using only the assigned dentifrice. Each subject was instructed to place an inch length of toothpaste on the wet toothbrush and to brush all surfaces of all teeth for at least one minute before expectorating. Each subject recorded his/her daily brushing in a diary which was provided. All assigned products were weighed before and after use by the investigator to assist in determining compliance. The diaries were checked at each visit by

a 3rd party who also distributed the assigned products. All patients attended all appointments on or close to day 56 with residual toothpastes. Recorded non-compliance with regard to dentifrice use was rare. Plaque was assessed using the Silness & Loe index (1964) and gingival condition by the Loe & Silness gingival index (1963). Both indices were determined at six sites; mesio-buccal, mid-buccal, disto-buccal, disto-lingual, mid-lingual and mesio-lingual on teeth 1–7 in each quadrant at 1 week pre-baseline, baseline, and at 2, 4, and 8 weeks thereafter.

Data analysis

All data were tested for Normality by plotting in ascending order against the corresponding Normal scores. A normal distribution was indicated by a reasonably straight line plot with no marked concavity or convexity. All data proved to be normally distributed. Paired *t*-tests were utilised for each treatment cell to determine if differences between readings at baseline and at

Table 1. Patient data

	Test group		Control group		Total	
	N	age \bar{x} (SD) (years)	N	age \bar{x} (SD) (years)	N	age \bar{x} (SD) (years)
female	13	42.6 \pm 11.38	12	43.9 \pm 8.24	25	43.2 \pm 9.81
male	7	40.3 \pm 4.88	8	43.8 \pm 3.84	15	42.1 \pm 4.57
mean	20	41.8 \pm 9.52	20	43.8 \pm 6.69	40	42.8 \pm 8.18

Test: silica-based.
Control: diatomaceous earth.
N: no. patients.

\bar{x} : mean.
SD: standard deviation.

scheduled examination times were statistically significant at the 95% confidence level. Similarly, at each time point, any differences between the dentifrices and their effects on plaque and gingival scores were tested for statistical significance by means of a two sample *t*-test. Confidence intervals were also calculated, and only probabilities of less than or equal to 0.05 were considered to indicate a significant difference between means. To avoid bias, all plaque and gingival scores were weighted for each individual to give the total as derived from either 28 teeth or 168 units as appropriate.

Results

Plaque accumulation

There was a slight increase in plaque accumulation in the first two weeks from baseline (Fig. 1), but relatively negligible change thereafter. The effect was identical in both groups. Paired *t*-tests demonstrated that any changes in the mean scores over the eight week period were negligible in terms of the

total possible score variation, and that there was no evidence that any apparent change in the mean score reflected an actual change in magnitude. Unpaired *t*-tests indicated no detectable differences between the groups at any time point.

Gingival status

There was a slight increase in Gingival Index in the first two weeks from baseline (Fig. 2), but relatively negligible change thereafter. GI tended to be higher in the control than in the test group. Paired *t*-tests demonstrated that any changes in mean gingival index over the 8-week period were negligible in terms of the total possible score variation, and there was no evidence that any apparent change in mean score reflected an actual change in magnitude. Unpaired *t*-tests indicated no detectable differences between the groups at any time point.

Discussion

Several investigators have suggested that plaque may play a role in the aeti-

ology of cervical dentinal hypersensitivity (CDH) (Everett et al. 1966, Grant et al. 1972, Chasens 1974, Schluger et al. 1977, Carranza 1984). Other work indicated that the level of plaque control is not a significant aetiological factor in CDH (Dowell et al. 1985), although several investigators assert the importance of good oral hygiene in the management of CDH (Grant et al. 1972, Chasens 1974, Schluger et al. 1977, Carranza 1984, Hovgaard et al. 1988). One of the problems, however, in comparing the effects of oral hygiene on CDH is that a variety of methods have been utilised to record the oral health status of participants. Toto et al. (1958) reported that oral hygiene ranged from poor to good, whereas Manochehr-Pour et al. (1984) reported that most participants showed an improvement in oral hygiene during the course of the trial, although no attempt was made to record plaque. More recent studies (Clark et al. 1985, Silverman 1986, Hovgaard et al. 1988, Salvato et al. 1989, Addy et al. 1990) attempted to measure plaque by partial or whole mouth recording, utilising the Greene & Vermillion (1960) or the Silness & Loe (1964) indices. One of the problems with the Greene & Vermillion index is that it is difficult to differentiate between bacterial plaque and other compounds once stained by a disclosing solution, resulting in an inaccurate assessment of plaque. In the present study, the Silness & Loe plaque index (1964) was used to record (by probe) plaque at six sites on all teeth excluding third molars.

Several desensitisation studies (Zinner et al. 1977, Gedalia et al. 1978, Silverman 1985, Addy et al. 1990) made no attempt to change the oral hygiene practices of participants during the trial, whereas Shapiro et al. (1970) and Hovgaard et al. (1988) attempted to carefully control hygiene procedures by instruction, reinforced at each visit and corrected if required (Shapiro et al. 1970). Other investigators (Gedalia et al. 1978, Clark et al. 1985), however, found, even when oral hygiene procedures were not changed prior to inclusion in desensitisation dentifrice trials, that there was little significant difference in plaque index between groups.

In the present study, no attempt was made to change the participants' oral hygiene, but all subjects received oral hygiene instruction and debridement prior to inclusion in the study, which may account for the relatively low

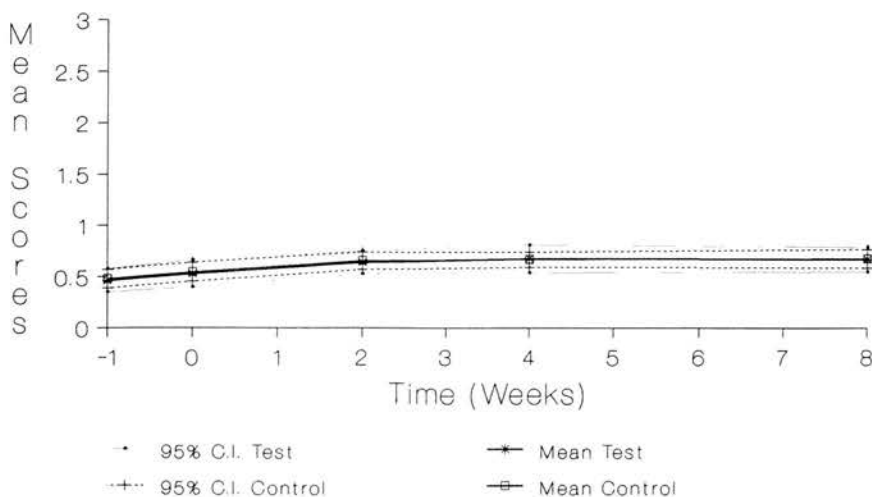


Fig. 1. Mean plaque index scores. Test: silica-based dentifrice. Control: diatomaceous earth-based dentifrice.

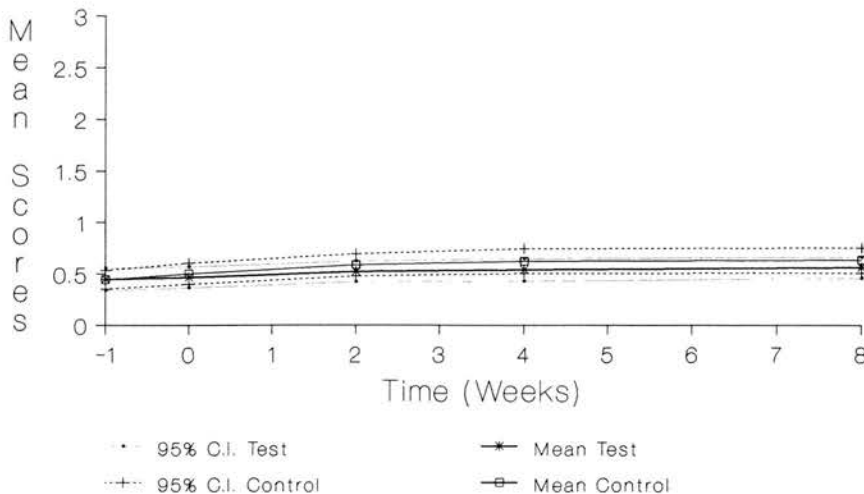


Fig. 2. Mean gingival index scores. Test: silica-based dentifrice. Control: diatomaceous earth-based dentifrice.

plaque and gingival index scores at the commencement of the study. The slight increase in plaque and gingival scores in the two weeks following baseline readings, and the levelling out of the mean values, may be explained by a slight relapse in oral hygiene following prebaseline treatment, and subsequent stabilised maintenance thereafter (Garcia-Godoy et al. 1990). It was also observed that no further change in PII and GI took place after 2 weeks. There was no evidence to suggest that any apparent change in the mean plaque and gingival scores reflected an actual change in magnitude. Neither plaque accumulation nor gingival condition significantly changed from baseline levels during the course of the study. The results of the present study appear to confirm the observations of Gedalia et al. (1978) and of Clark et al. (1985) in that there was little or no change between the two groups in plaque scores. Indeed, the plaque effect was identical in both test and control groups. In summary, there was no evidence to suggest that SCH dentifrices increased plaque accumulation, or that the abrasivity of the desensitizing dentifrice affected the level of plaque.

The results of the present study, therefore, do not support the conclusions of previous studies which indicated that SCH dentifrices increased plaque accumulation. It was notable that neither SCH dentifrices had any clinically significant effect per se on plaque or gingival condition.

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Zusammenfassung

Die Wirkung von Strontiumchlorid-Hexahydrat-haltigen Zahnpasten auf die Plaqueakkumulation und Gingivaentzündung

Im Rahmen einer randomisierten klinischen Doppelblindstudie, die sich über zwei Monate erstreckte, wurden zwei Strontiumchlorid-Hexahydrat (SHC)-haltige Zahnpasten, die sich durch nichts, außer den verwendeten Abrasivstoffen, entweder Diatomeenerde oder Silikate, unterschieden, hinsichtlich ihrer Wirkung auf die Plaqueakkumulation und Gingivaentzündung verglichen. Vor der Teilnahme an der Studie wurde nichts zur Veränderung der Mundhygiene des Patienten unternommen. Die Plaque wurde mit dem Silness & Loe und der Gingivazustand mit dem Loe & Silness Index GI gemessen. Zwei Wochen nach der Eingangsuntersuchung ergab sich eine leichte, aber nicht signifikante Zunahme in der Plaqueakkumulation und danach nur vernachlässigbare Veränderungen, die in beiden Gruppen identisch waren. Der Gingivazustand zeigte eine ähnliche Veränderung des Indexwertes nach der Eingangsuntersuchung, trotz der leicht höheren Tendenz in der Diatomeenerden-Gruppe. Die Ergebnisse unterstützen nicht die Schlussfolgerungen früherer Studien, die zeigten, daß SHC-Zahnpasten die Plaqueakkumulation erhöhten. Im Verlauf der Studie veränderte sich weder die Plaqueakkumulation noch der

Gingivazustand signifikant bezüglich der Werte bei der Eingangsuntersuchung.

Résumé

Effet de dentifrices au chlorure de strontium hexahydrate sur l'accumulation de la plaque et sur l'inflammation gingivale

Au cours d'une étude clinique randomisée à double insu en parallèle faite sur 2 mois, les effets de 2 dentifrices contenant du chlorure de strontium hexahydrate (SCH) sur l'accumulation de la plaque et l'inflammation gingivale ont été comparés. Ces 2 dentifrices avaient la même composition, à l'exception des éléments assurant l'abrasion, basée dans l'un sur une terre diatomée, dans l'autre sur la silice. Aucun effort n'a été fait pour modifier les pratiques d'hygiène bucco-dentaire des patients avant leur participation à cette étude. La plaque a été enregistrée à l'aide de l'indice de Silness & Loe et l'état gingival par l'indice gingival GI de Silness. On notait une faible augmentation de l'accumulation de plaque deux semaines après le début, mais cette augmentation n'était pas significative et il ne se produisait ensuite que des changements relativement négligeables, l'effet étant identique dans les deux groupes. De même, l'indice de l'état gingival présentait un faible changement par rapport à celui du début; il tendait cependant à être légèrement plus élevé dans le groupe de la terre diatomée. Ces résultats ne confortent pas les conclusions d'études antérieures indiquant que les dentifrices au SCH augmentaient l'accumulation de plaque. Ni l'accumulation de plaque ni l'état gingival ne se modifiaient significativement au cours de cette étude par rapport aux niveaux initiaux.

References

- Addy, M., Mostafa, P. & Newcombe, R. G. (1990) Effect on plaque of five toothpastes used in the treatment of Dentin Hypersensitivity. *Clinical Preventive Dentistry* **12**, 28-33.
- Blitzer, B. (1967) A consideration of the possible causes of dental hypersensitivity. Treatment by a strontium-ion dentifrice. *Periodontics* **5**, 318-321.
- Carranza, F. A. (1984) In: *Glickman's clinical periodontology*, 6th edition. W. B. Saunders Company, Philadelphia, pp. 384.
- Carrasco, -P. H. (1971) Strontium chloride toothpaste. Effectiveness as related to duration of use. *Pharmacol. Ther. Dent.* **1**, 209-215.
- Chasens, A. I. (1974) The management of pain and sensitivity. In: *Mechanisms of pain and sensitivity of teeth and supporting tissues*. Chasens, A. I. & Kaslick, R. S. (eds.) Fairleigh Dickinson University School of Dentistry and American Academy of Oral Medicine, pp. 12-15.
- Clark, D. C., Hanley, J. A., Geoghegan, S. & Vinet, D. (1985). The effectiveness of a fluoride varnish and a desensitizing tooth-

- paste in treating dentin hypersensitivity. *Journal of Periodontal Research* **20**, 212–219.
- Collins, J. F., Gingold, J., Stanley, H. & Simring, M. (1984) Reducing dentinal hypersensitivity with strontium chloride and potassium nitrate. *General Dentistry* **32**, 40–43.
- Dowell, P., Addy, M. & Dummer, P. (1985) Dentine Hypersensitivity: Aetiology, Differential Diagnosis and Management. *British Dental Journal* **158**, 92–96.
- Everett, F. G., Hall, W. B. & Phatak, N. M. (1966) Treatment of hypersensitive dentine. *Journal of Oral Therapeutics and Pharmacology* **2**, 300–310.
- Garcia-Godoy, F., Garcia-Godoy, F., De Vizio, W., Volpe, A. R., Ferlauto, R. J. & Miller, J. M. (1990) Effect of a triclosan/copolymer/fluoride dentifrice on plaque formation and gingivitis: A 7-month clinical study. *American Journal of Dentistry* **3**, 15–26.
- Gedalia, I., Brayer, L., Kalter, N., Richter, M. & Stabholz, A. (1978) The effect of fluoride and strontium application on dentin: *in vivo* and *in vitro* studies. *Journal of Periodontology* **49**, 269–272.
- Gillam, D. G., Newman, H. N., Bulman, J. S. & Davies, E. H. (1991) Dentifrice abrasivity and cervical dentinal hypersensitivity. *Journal of Dental Research* **70**, 205 (abstr.).
- Grant, D. A., Stern, I. B. & Everett, F. G. (Eds.) (1972) In: *Orban's periodontics*, 4th edition. C. V. Mosby Company, Saint Louis, pp. 414–416.
- Greene, J. C. & Vermillion, J. R. (1960) The oral hygiene index: A method of classifying oral hygiene status. *Journal of the American Dental Association* **61**, 172–178.
- Hernandez, F., Mohammed, C. O., Shannon, I., Volpe, A. & King, W. (1972) Clinical study evaluating the desensitizing effect and duration of two commercially available dentifrices. *Journal of Periodontology* **43**, 367–372.
- Hovgaard, O., Larsen, M. J. & Fejerskov, O. (1988) Hypersensitive Root Surfaces – Effect of Oral Hygiene and Topical Application of NaF and Ca(OH)₂. *Journal of Dental Research* **67**, 1734 (abstr.).
- Jackson, R. J., Duke, S. A. & Wicks, M. A. (1989) The evaluation of two dentifrices formulated for the treatment of dentine hypersensitivity. *Journal of Dental Research* **68**, 603 (abstr.).
- Löe, H. & Silness, J. (1963) Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odontologica Scandinavica* **21**, 533–551.
- Manochehr-Pour, M., Bhat, M. & Bissada, N. (1984) Clinical evaluation of two potassium nitrate toothpastes for the treatment of dental hypersensitivity. *Periodontal Case Reports* **6**, 25–30.
- Minkoff, S. & Axelrod, S. (1987) Efficacy of strontium chloride in dental hypersensitivity. *Journal of Periodontology* **58**, 470–474.
- Salvato, A., Clark, G., Curro, F. A. (1989) Clinical efficacy of Sensodyne-F as a desensitizing dentifrice. *Journal of Dental Research* **68**, 839 (abstr.).
- Schluger, S., Yuodelis, R. A. & Page, R. C. (1977) *Periodontal disease*. Lea & Febiger, Philadelphia, pp. 769–770.
- Shapiro, W. B., Kaslick, R. S., Chasens, A. I. & Weinstein, D. (1970) Controlled clinical comparison between a strontium chloride and a sodium monofluorophosphate toothpaste in diminishing root hypersensitivity. *Journal of Periodontology* **41**, 523–525.
- Shapiro, W. B., Kaslick, R. S. & Chasens, A. I. (1970) The effect of a strontium chloride toothpaste on root sensitivity in a controlled clinical study. *Journal of Periodontology* **41**, 702–703.
- Silness, J. & Löe, H. (1964) Periodontal disease in pregnancy. (II). Correlation between oral hygiene and periodontal condition. *Acta Odontologica Scandinavica* **22**, 121–135.
- Silverman, G. (1985) The sensitivity-reducing effect of brushing with a potassium nitrate-sodium monofluorophosphate dentifrice. *Compendium of Continuing Education in Dentistry* **6**, 131–136.
- Silverman, G. (1986) Data on file. Stafford-Miller Limited, Hatfield, Herts, U.K.
- Toto, P. D., Staffileno, H. & Gargiulo, A. W. (1958) A clinical evaluation of a desensitizing toothpaste. *Journal of Periodontology* **29**, 192–194.
- Uchida, A., Wakano, Y., Fukuyama, O., Miki, T., Iwayama, Y. & Okada, H. (1980) Controlled clinical evaluation of 10% strontium chloride dentifrice in treatment of dentin hypersensitivity following periodontal surgery. *Journal of Periodontology* **51**, 578–581.
- Zinner, D. D., Duany, L. F. & Lutz, H. J. (1977) A new desensitizing dentifrice: preliminary report. *Journal of the American Dental Association* **95**, 982–985.

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Iontophoresis in the Treatment of Cervical Dentinal Sensitivity — A Review

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Introduction

Definition. According to Scott (1962), electrophoresis, iontophoresis, ionization, cataphoresis, anaphoresis, electrolytic medication, and other names have been used to designate a means of applying medications with the assistance of a small electric current. More recently, iontophoresis has been described as a method of facilitating the transfer of ions by means of an electrical potential into soft or hard tissues of the body for therapeutic purposes (Walton *et al.* 1979, Pashley 1985).

Iontophoresis of fluoride for the treatment of cervical dentinal sensitivity, however, has been controversial. Several investigators (Murphy *et al.* 1973, Gangarosa and Park 1978, Gangarosa *et al.* 1978, 1989, Gangarosa 1981, Carlo *et al.* 1982, Lutins *et al.* 1984, Gangarosa and McRae 1985, Klaus and Gangarosa 1986, Kern *et al.* 1989) have reported successful desensitization of dentine by this method, while others (Minkov *et al.* 1975, Schaeffer *et al.* 1971, Brough *et al.* 1985) have reported conflicting results attributable to error or to a lack of standardization in the basic tech-

nique of iontophoresis used in these studies (Gangarosa and Park 1978, Gangarosa 1986).

Iontophoresis, however, is not new. One of its first recorded uses was by Richardson (1859), cited by Morton (1896), who used chloroform and aconite to anesthetize a dog's leg. Morton (1896) recommended the use of a cocaine and guaiacol solution in order to anesthetize sensitive dentine, dental pulp, and soft tissues. Studies have also shown that it is possible for radioactive ions, including those of iodide, calcium, and sodium, to penetrate dentine by iontophoresis (Sausen 1955, Stowell *et al.* 1961, Pashley *et al.* 1978). Investigators have also observed that there is an increased fluoride uptake in exposed root dentine following topical application of two percent sodium fluoride, and that the fluoride ion has a marked affinity for calcium, which in turn may react in fluids to form CaF_2 (Ehrlich *et al.* 1975). Zadok *et al.* (1976) demonstrated that fluoride iontophoresis, when compared with topical fluoride alone, resulted in an increase in the uptake of the fluoride ion into the dentine without any adverse pulpal changes.

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Mechanism of Fluoride Iontophoresis

The exact mechanism of fluoride ion-

tophoresis is not known, although several hypotheses have been proposed. Lefkowitz (1960), Scott (1963), Lefkowitz *et al.* (1963), and Murthy *et al.* (1973) suggested that the desensitization of dentine was the result of the formation of secondary dentine by the electrical current (iontophoresis). Gangarosa and Park (1978) proposed that iontophoresis produced paresthesia by altering the sensory mechanism of pain conduction. A third possible mechanism, based on the hydrodynamic theory (Brannstrom 1962, 1963, Brannstrom and Astrom 1972), hypothesized that fluoride iontophoresis may increase the concentration and depth of penetration of fluoride ions in dentinal tubules, which in turn may cause a microprecipitation of calcium fluoride, thereby occluding the tubules and reducing the conduction of hydrodynamically mediated stimuli (Pashley 1985, Kern *et al.* 1989).

Possible Hypotheses

1. Induction of secondary dentine formation by iontophoresis

Lefkowitz and co-workers (1960, 1963) reported on the pulpal response to one percent NaF iontophoresis and claimed that the electric current was responsible for inducing the formation of secondary dentine without causing permanent pulpal damage, irrespective of whether sodium fluoride or saliva was applied. This indicated that the current and not the fluoride ion was the effective desensitization agent, an assertion supported by Scott (1962), Schaeffer *et al.* (1971), and Murthy *et al.* (1973). Scott (1962) also reported that one percent sodium fluoride iontophoresis disrupted the odontoblast layer under cut dentine during a two-week period, caused a delayed pulpal recovery and a gross deposit of secondary dentine, but concluded iontophoresis of one percent sodium fluoride for 1mA/minute caused no permanent pulpal damage. Seltzer and Bender (1975), however, questioned the rapidity of secondary dentine deposition, and Walton *et*

al. (1979), while agreeing with Lefkowitz and Scott's observation that iontophoresis caused no permanent pulpal damage, failed to find any evidence of secondary dentine formation after 7 or 56 days following application of one percent sodium fluoride by iontophoresis at therapeutic (0.5mA/2min.) or five times therapeutic levels on exposed dentine in dogs. The disruption in the odontoblast layer noted by Sauser (1955) and Scott (1962) was not observed by Walton and Eisenmann (1975) or Walton *et al.* (1979).

Results appear to vary according to the experimental models and the choice of site and its preparation. Both Scott and Lefkowitz prepared buccal cavities on human teeth which were later extracted, whereas Walton *et al.* (1979) utilized dog root surfaces denuded of cementum. Lefkowitz's conclusions have been disputed (Walton *et al.* 1979) since the study was incompletely controlled in that untreated teeth from the same subjects were not compared. The studies did not use hard tissue markers to indicate the amount of dentine formation prior to and after the experiments. Scott (1962) reported that fluoride iontophoresis on the cavity floor disrupted the odontoblast layer. However, it was observed that controls (cavity preparation only) also demonstrated pulpal disruption, but to a lesser degree. Furthermore, there was no way of differentiating the effect of iontophoresis from the effect of cavity preparation *per se*; whereas in the Walton *et al.* (1979) study, the investigators, by utilizing both positive and negative controls, demonstrated that removing surface layers of dentine from the root caused no effect on the odontoblasts or pulp. Walton did not rule out changes at the molecular level, but concluded that desensitization was not due to the alteration of the odontoblast layer or the formation of secondary dentine, but to tubular occlusion. Gangarosa and Park (1978) suggested that the observed clinical effect of fluoride iontophoresis is immediate relief of sensitivity and suggested that

the formation of secondary dentine as proposed by Lefkowitz could not account for this.

2. *Induction of paresthesia on odontoblastic processes by iontophoresis* (Alteration of the sensory mechanism of pain conduction)

The second hypothesis is that the electric current may either produce paresthesia by a direct effect on the odontoblastic processes (Brough *et al.* 1985) or by alteration of the sensory mechanism of pain conduction (Gangarosa and Park 1978), but evidence at present does not support such a hypothesis. Gangarosa and Park (1978) suggested that since desensitization of dentine is a lengthy procedure, one would have to suggest either that the paresthesia produced is long-acting or that an alternative mechanism is responsible. Gangarosa *et al.* (1977), however, reported that changes in nerve conduction as a result of direct current application were temporary, the nerves recovering immediately after the removal of current. Walton *et al.* (1979) did not observe any intracellular changes in the sensory nerves or in cells responsible for interference with nerve conduction. These changes were not sufficient to cause any observable alteration of myelinated or unmyelinated nerves in the central pulp.

3. *Increased fluoride ion concentration and depth of ion penetration into dentine induced by iontophoresis* (Tubule occlusion)

The third hypothesis proposed is based on the hydrodynamic theory (Brannstrom 1962, 1963, Brannstrom and Astrom 1972). Several studies have suggested that the fluoride ion concentration in dentinal tubules is increased by iontophoresis and that the increased concentration of ions causes a microprecipitation of calcium and fluoride which serve to occlude the tubules, thereby preventing the conductance of hydrodynamically mediated stimuli (Gangarosa *et al.* 1985, Kern *et al.* 1989). Wilson *et al.* (1984) demonstrated that ion-

tophoresis caused a significantly greater depth of penetration of the fluoride ion into the dentine than topical application alone. Once inside the dentinal fluid, they reasoned that the fluoride ion is available to combine with the calcium ion to form an insoluble precipitate of calcium fluoride which can physically occlude the tubule. Greenhill and Pashley (1981) demonstrated that iontophoresis of two percent sodium fluoride induced a greater percentage fall in hydraulic conductance (L_p) than non-iontophored sodium fluoride and suggested that the negative ion was able to move down the electrical gradient created by the apparatus, thereby enabling deeper penetration of the fluoride ion into the tubule than would occur by diffusion alone, thus increasing the probability of tubule occlusion (Wilson *et al.* 1984).

More research, however, is needed, not only to select the best active agent for iontophoresis, but also to elucidate the mechanism underlying its effect. To date, desensitization by tubule occlusion appears to be the most probable mechanism, although other mechanisms may still be implicated (Walton *et al.* 1979, Pashley 1985).

Clinical Evidence

Most studies have reported that fluoride iontophoresis is a safe and effective method of treating cervical dentinal sensitivity. According to Gangarosa and Park (1978) and Gangarosa (1986) the procedure of iontophoresis, if successfully adhered to, will provide an immediate effect in reducing sensitivity which may gradually diminish over time (Kern *et al.* 1989).

Several investigators, however, have found iontophoresis a costly, time-consuming and technique-demanding procedure which is extremely operator sensitive (Pashley 1985). Many of the studies evaluating iontophoresis have lacked suitable controls and standardization of the procedure, and in consequence it is difficult to differentiate the topical effects of the active agent from any additional ion-

tophoretic effect, as well as possible associated placebo and non-placebo effects (Karlsson and Penney 1975, Peden 1977, Addy and Dowell 1983).

Investigators have also utilized a variety of electrolytes and modes of application. Some studies have used iontophoretic toothbrushes, either with stannous or sodium fluoride in solution or dentifrice form (Collins 1962, Siemon 1962, Jensen 1964, Schaeffer *et al.* 1971, Johnson *et al.* 1982). Others have used an iontophoresis device (Siemon 1960, Manning 1961, Gangarosa and Park 1978, Gangarosa *et al.* 1978, Carlo *et al.* 1982, Lutins *et al.* 1984, Klaus and Gangarosa 1986, Gangarosa *et al.* 1989, Kern *et al.* 1989) or a tray system (Gangarosa 1981).

Various electrolytes have been used: saliva, sodium chloride, stannous and sodium fluorides (Murthy *et al.* 1973, Gangarosa and Park 1978, Gangarosa *et al.* 1978, 1989, Brough *et al.* 1985, Kern *et al.* 1989). Most have used a negative electrode and have reported reductions in sensitivity, although the studies which have utilized a positive electrode have reported confusing and occasionally negative results (Collins 1962, Jensen 1964, Schaeffer *et al.* 1971, Minkov *et al.* 1975, Johnson *et al.* 1982, Brough *et al.* 1985). This apparent discrepancy may derive from the use of a positive electrode which resists the penetration of the negative fluoride ion (Gangarosa and Park 1978, Gangarosa 1984).

Other possible sources of error include the choice of electrolyte and the fact that saliva contains sodium chloride which may compete with the fluoride ion for the current. Brough *et al.* (1985) failed to report an immediate effect in relief of sensitivity, although there was a slight decrease in sensitivity over time. Gangarosa (1986) suggested that the possible reason for this was a failure to comply with the iontophoresis procedure, as one would have expected to observe an immediate result. The use of rubber dam may have caused a loss of current flow, and the use of saliva as one of the electrolytes, as well as selection

of patients who had had recent periodontal surgery, may have contributed to the negative result.

Conclusions

Most studies on the use of sodium fluoride with iontophoresis have good short-term results, although only a few have reported long-term results (Klaus and Gangarosa 1986, Gangarosa *et al.* 1989, Kern *et al.* 1989). There is still a need for evaluation of the effectiveness of fluoride iontophoresis over time using adequate controls and suitable test methods, which are both quantifiable and reproducible, as recommended by the American Dental Association, Council on Dental Therapeutics (1986).

References

- Addy, M., and P. Dowell: Dentine hypersensitivity — A Review. Clinical and in vitro evaluation of treatment agents, *J. Clin. Periodont.*, 10:351, 1983.
- Brannstrom, M.: The elicitation of pain in human dentine and pulp by chemical stimuli, *Arch. Oral Biol.*, 7:59, 1962.
- Brannstrom, M.: A hydrodynamic mechanism in the transmission of pain-producing stimuli through the dentine, Anderson, D.J., ed. Oxford, Pergamon Press, 73, 1963.
- Brannstrom, M., and A. Astrom: The hydrodynamics of dentine: its possible relationship to dentinal pain, *Int. Dent. J.*, 22:219, 1972.
- Brough, K.M., D.M. Anderson, J. Love, and P.R. Overman: The effectiveness of iontophoresis in reducing dentin hypersensitivity, *J.A.D.A.*, 111:761, 1985.
- Carlo, G.T., S.G. Ciano, and S.K. Seyrek: An evaluation of iontophoretic application of fluoride for tooth desensitization, *J.A.D.A.*, 105:452, 1982.
- Collins, E.M.: Desensitization of hypersensitive teeth, *Dent. Dig.*, 68:360, 1962.
- Council on Dental Therapeutics, ADA Ad Hoc Advisory Committee: Recommendations for evaluating agents for the reduction of dentinal hypersensitivity, *J.A.D.A.*, 112:709, 1986.
- Ehrlich, J., N. Hockman, I. Gedalia, and M. Tal: Residual fluoride concentrations and Scanning Electron Microscope examination of root surfaces of human teeth after topical application of fluoride in vivo, *J. Dent. Res.*, 54:897, 1975.
- Gangarosa, L.P.: Iontophoretic application of fluoride by tray techniques for desensitization of multiple teeth, *J.A.D.A.*, 102:50, 1981.
- Gangarosa, L.P.: Letter to the Editors, *J. Periodont.*, 55:252, 1984.
- Gangarosa, L.P.: Letters to the Editor, *J.A.D.A.*, 112:808, 1986.
- Gangarosa, L.P., N.H. Park, and G. King: Iontophoresis of lidocaine into frog sciatic nerve fibers, *Life Sci.*, 21:885, 1977.
- Gangarosa, L.P., and N.H. Park: Practical considerations in iontophoresis of fluoride for desensitizing dentin, *J. Prost. Dent.*, 39:173, 1978.
- Gangarosa, L.P., G. Heuer, N. Clark, B. Haynes, C. Little, G.

- Baker, and A. Smith: Desensitizing hypersensitive dentin by iontophoresis and fluoride, *N.Y. Dent. J.*, 44:92, 1978.
- Gangarosa, L.P., M. Gado, and R.S. Baratz: Occlusion of dentinal tubules by iontophoretic application of sodium fluoride, *J. Dent. Res.* (abstracts), 64:309, 1985.
- Gangarosa, L.P., and K. McRae: Fluoride iontophoresis as an aid in the diagnosis and treatment of reversible pulpitis. A case study, *Comp. Cont. Ed.*, 6:696, 1985.
- Gangarosa, L.P., A.L. Buettner, W.P. Baker, B.K. Buettner, and W.O. Thompson: Double-blind evaluation of duration of dentin sensitivity reduction by fluoride iontophoresis, *Gen. Dent.*, July/August:316, 1989.
- Greenhill, J.D., and D.H. Pashley: The effects of desensitizing agents on the hydraulic conductance of human dentin in vitro, *J. Dent. Res.*, 60:686, 1981.
- Johnson, R.H., B.J. Zulqar-Nain, and J.J. Koval: The effectiveness of an electro-ionizing toothbrush in the control of dentinal hypersensitivity, *J. Periodont.*, 53:353, 1982.
- Karlsson, U.L., and D.A. Penney: Natural desensitization of exposed tooth roots in dogs, *J. Dent. Res.*, 54:982, 1975.
- Kern, D.A., M.J. McQuade, M.J. Scheidt, B. Hanson, and T.E. Van Dyke: Effectiveness of sodium fluoride on tooth hypersensitivity with or without iontophoresis, *J. Periodont.*, 60:386, 1989.
- Klaus, B., and L.P. Gangarosa: The long-term effect of sodium fluoride iontophoresis for treatment of hypersensitive dentin, *Quintessenz*, 38:81, 1986.
- Lefkowitz, W.: Pulp response to ionization, *J. Prosth. Dent.*, 12:966, 1960.
- Lefkowitz, W., B.A. Burdick, and D.L. Moore: Desensitization of dentin by bioelectric induction of secondary dentin, *J. Prosth. Dent.*, 13:940, 1963.
- Lutins, N.L., G.W. Greco, and W.T. McFall: Effectiveness of sodium fluoride on tooth hypersensitivity with and without iontophoresis, *J. Periodont.*, 55:285, 1984.
- Manning, M.W.: New approach to desensitization of cervical dentin, *Dent. Survey*, 37:731, 1961.
- Minkov, B., I. Marmari, I. Gedalia, and A. Garfunkel: The effectiveness of sodium fluoride treatment with and without iontophoresis on the reduction of hypersensitive dentin, *J. Periodont.*, 46:246, 1975.
- Morton, W.J.: Guaiacol-cocaine cataphoresis and local anesthesia, *Dent. Cosmos*, 38:48, 1896.
- Morton, W.J.: Cataphonic medication in the mouth, *Dent. Cosmos*, 38:400, 1896.
- Murthy, K.S., S.T. Talim., and I. Singh: A comparative evaluation of topical and iontophoresis of sodium fluoride for desensitization of hypersensitive dentin, *Oral Surg.*, 36:448, 1973.
- Pashley, D.H.: Strategies for clinical evaluation of drugs and/or devices for the alleviation of hypersensitive dentin, In: Rowe, N.H. (ed). *Proceedings of Symposium on Hypersensitive Dentin: Origin and Management*. University of Michigan, 65-88, 1985.
- Pashley, D.H., M.J. Livingston, and W.C. Outhwaite: Dentin permeability. Changes produced by iontophoresis, *J. Dent. Res.*, 57:77, 1978.
- Peden, J.W.: Dental hypersensitivity: *J. Western Soc. Periodont./Periodont. Abs.*, 25:75, 1977.
- Sausen, R.E.: Electrophoresis in dentin with radioactive calcium, *J. Dent. Res.*, 34:12, 1955.
- Scott, M.: Reduction of sensitivity by electrophoresis, *J. Dent. Child.*, 19:225, 1962.
- Schaeffer, M.L., D. Bixler, and Y. Pao-Lo: The effectiveness of iontophoresis in reducing cervical hypersensitivity, *J. Periodont.*, 42:695, 1971.
- Seltzer, S., and I. Bender: Desensitizing and remineralizing agents. In: *The Dental Pulp*, ed. 2. Philadelphia, J.B. Lippincott Company, 202-210, 1975.
- Siemon, W.H.: A new approach to solving the problem of hypersensitivity and post-operative distress in dentin and cementum, *J. Conn. Dent. Assoc.*, 34:5, 1960.
- Siemon, W.H.: Observations on the value of iontophoretic procedures in dentistry, *Dent. Dig.*, 68:172, 1962.
- Stowell, E.C., J.B. Tayler, and W.W. Wainwright: Ion penetration through teeth as influenced by an electrostatic field, *J. Dent. Res.*, 40:739, 1961.
- Walton, R.E., and D.R. Eisenmann: Ultrastructural examination of dentine formation in rat incisors following multiple fluoride injections, *Arch. Oral Biol.*, 20:485, 1975.
- Walton, R.E., L.A. Leonard, M. Sharawy, and L.P. Gangarosa: Effects on pulp and dentin of iontophoresis of sodium fluoride on exposed roots in dogs, *Oral Surg.*, 48:545, 1979.
- Wilson, J.M., B.W. Fry, R.E. Walton, and L.P. Gangarosa: Fluoride levels in dentin after iontophoresis of 2% NaF, *J. Dent. Res.*, 63:897, 1984.
- Zadok, J., I. Gedalia, J. Weinmann, and L. Daphni: Fluoride uptake by root dentin after immersion in 2% NaF solution with iontophoresis, *J. Dent. Res.*, 55:310, 1976.

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Assessment of pain in cervical dentinal sensitivity studies

A review

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Abstract. Traditionally cervical dentinal sensitivity (CDS) has been evaluated mainly subjectively on the basis of the individual patient's subjective response, e.g., in the form of verbal rating and visual analogue scales and questionnaires. The stimuli used for evaluating this response can be grouped into 4 main categories: mechanical, chemical, electrical and thermal. This review of the literature, however, indicates that there are problems in evaluating patient subjective response to these various test stimuli used in the assessment and treatment of CDS. Opinions also vary as to the reliability of some of these methods of assessment, although recently, efforts have been made to develop controlled reproducible stimuli more suited to the evaluation of CDS. Currently no single method of eliciting and assessing CDS may be considered ideal. Further research is required to evaluate suitable methodology for the quantification of realistic test stimuli under controlled clinical conditions, whereby the subjective response may be objectively measured by the investigator.

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Cervical dentinal sensitivity (CDS) has been defined as pain arising from exposed dentine, typically in response to chemical, thermal, tactile or osmotic stimuli, which cannot be explained as arising from other forms of dental defect or pathology (Addy et al. 1985). Traditionally, CDS has been evaluated mainly subjectively on the basis of the individual patient's subjective response, for example, in the form of verbal rating and visual analogue scales and questionnaires. The stimuli can be grouped into four main categories: mechanical, chemical, electrical and thermal. The method and interpretation of pain assessment elicited from such stimuli, however, is open to question and interpretation. Furthermore, the subjective nature of the response and variability in patient ability to express a given response may also complicate assessment. Currently no single method of eliciting and assessing CDS may be considered ideal.

Reproducibility of the stimulus

Variability in both stimuli and response to individual types of stimulus constitute major deficiencies in current efforts to monitor and evaluate CDS. In order to overcome such deficiencies the American Dental Association (1986) recommended the following study design features.

(1) The test data should be both quantifiable and reproducible.

(2) A critical evaluation must be made of all subjective responses. The threshold of response should be established, preferably quantified, and correlated to a clinically definable intensity. It is also recognised that the threshold is a range and not a point.

(3) The relationship between the experimental stimulus and the defined area of hypersensitivity must be established by properly controlled clinical research.

(4) There should be no commitment to

a specific form of stimulus. If more than one stimulus is used, then these stimuli should be reproducible and interference between them must be minimised.

(5) Appropriate statistics should be used, and these should be justified according to the experimental design.

In addition to these study design features, the committee recommended the use of a variable stimulus level-fixed threshold response as opposed to the earlier method of fixed stimulus level-variable response for the evaluation of CDS (Kanapka 1990).

Subject Assessment

Pain has been described as a subjective and multidimensional experience (Melzack 1973, 1975, McGrath 1986). The diversity of the pain experience, however, explains why it has been impossible to provide a satisfactory definition of the word, pain. Melzack & Wall

(1988), suggest the reason for this is that the word pain represents a category of experiences having different causes and characterised by different qualities, varying along a number of sensory, affective and evaluative dimensions. The perception of pain is based on a number of variables including the significance of pain, individual personality, psychological factors, cultural attitudes, anticipation of pain and the degree of apprehension (Mumford 1973).

Problems in evaluating the effectiveness of a desensitizing agent in a clinical trial may, therefore, derive from a lack of predictable, reliable and reproducible methodology for evaluating the subjective response of the patient, which can be further modified by social, cultural, psychological and situational factors (Ash 1986, McGrath 1986).

Verbal and non-verbal (numerical) scales as well as questionnaires such as the McGill Pain Questionnaire (MPQ) have been used to provide both qualitative and quantitative information on the subjective nature of pain following an evoked response from a painful stimulus.

According to Clark & Troullos (1990), qualitative evaluation of the subjective response in CDS clinical trials, using verbal descriptors provided by the patients themselves to describe pain, has not been documented. The patients' quantitative assessment of their own overall perception of pain associated with CDS, however, has been evaluated in clinical studies (Brough et al. 1985, Silverman 1985, Clark et al. 1987, Orchardson & Collins 1987, Minkoff & Axelrod 1987, Gillam et al. 1992a, b). Patients were asked to rate their own perception of overall sensitivity to hot/cold food and drink, air, toothbrushing and sweet and sour food as experienced during everyday routine. They reported using either a verbal rating scale (VRS) or a visual analogue scale (VAS). McGill word group descriptors, part of the MPQ, may also be used for this purpose.

Evaluation of the subjective response following tactile, thermal, and electrical stimuli may also be recorded by the patient in the same manner.

Verbal rating scales ((VRS)

Keele (1948) described a 4-point scale grading pain as slight, moderate, severe and agonising. This simple descriptive

pain scale has been modified and a typical VRS may look like the following:

- 0 = no discomfort
- 1 = mild discomfort
- 2 = marked discomfort
- 3 = marked discomfort that lasted more than 10 s

VRS offer a restrictive choice of words which may not represent the pain experience with significant precision for all patients (Huskiison 1974, Clark & Troullos 1990). The mathematical interpretation of the scoring system has also been challenged, in that the scores are often arbitrarily assigned numerical values, and the assigned scores are then analysed as if these numbers reflected true quantitative differences in pain, rather than simple qualitative differences (McGrath 1986).

Visual analogue scales (VAS)

A visual analogue scale is a line 10 cm in length, the extremes of the line representing the limits of pain a patient might experience from an external stimulus (no pain at one end and severe pain or discomfort at the other end of the line). Patients are asked to place a mark on the 10 cm line which indicates the intensity of their current level of sensitivity or discomfort following application of test stimuli. VAS pain intensity can be shown either as an absolute score value or as a % of the maximum. The validity and reliability of the VAS for measuring both experimental and clinical pain has been demonstrated by several investigators. Clark & Troullos (1990) reported that once the VAS procedure is properly explained to patients, it is simple to understand and suitable for use in the evaluation of stimuli response in CDS dentifrice studies. Several investigators have compared the VAS with other pain scales and the results indicate that the VAS correlates well with these methods and appears to be more sensitive in discriminating between various treatments and changes in pain intensity (Ekowski et al. 1972, Joyce et al. 1975, Ohnhaus & Adler 1975). Downie et al. (1978) reported that numerical rating scales (0–10) performed better than both four point descriptive scales and a continuous (visual analogue) scale. Scott & Huskiison (1976) demonstrated that graphic rating scales which are VAS, with descriptive terms placed at intervals along a 10 cm line, may have the advantage of helping the patient decide the position of his score, especially

in the absence of previous experience of pain measurement procedures, as well as enabling different subjects to record the same degree of severity of pain in the same position. These investigators concluded that this type of rating provided the best available method for measuring pain or pain relief. One objection to the graphic rating scale is that the words underneath the scale may induce a higher density of clustering of responses close to them (Seymour 1982).

Although Seymour (1982) questioned the validity of any postulated advantage to be gained by using the graphic rating scale as opposed to the plain 10 cm VAS, it is apparent that the VAS can only give a unidimensional assessment of pain, and as such cannot distinguish between the sensory, intensity and affective (unpleasantness) aspects of pain.

McGill pain questionnaire (MPQ)

One of the first verbal tests which addressed the multidimensional nature of pain was the MPQ (Melzack 1975). The MPQ has been used to evaluate a variety of painful dental conditions including CDS. One limitation in clinical trials, however, is its complexity of vocabulary. The patient is shown 20 sets of words and asked to select a word from each set which best describes present pain experience. Each set contains up to 6 words in ascending order of severity. 10 of the word sets describe sensory qualities, 5 are affective descriptor sets, and 1 set describes the evaluative dimension of pain; the remaining 4 sets are classified as miscellaneous although they appear to be predominantly sensory. The number of words chosen provides one index (NWC), and since the words within each group set have been arranged in rank order, one can add up the total rank of all words chosen to obtain a pain rating Index (PRI). Additional information regarding the type of medication used for the pain, pain location and comparison of the present pain to previous pain experience may be obtained using the unabridged version of the MPQ. One of the advantages of the MPQ is that it provides additional data on both the qualitative and quantitative aspects of pain. Limitations of the MPQ, may, however, preclude its use in CDS studies, as it is more time-consuming to administer compared to VAS and category scale procedures. The test may

reflect, in part, the vocabulary limitations of the patient as well as the nature of pain *per se*. There may also be cultural differences in language habits which could be confounded with differences in pain expression. Patients are forced to give more consideration to the sensory aspects of pain rather than the affective or evaluative aspects in the test procedure (Chapman et al. 1985). Several investigators (Hall et al. 1986, Zakrzewska & Feinmann 1990) have reported that the MPQ is useful in diagnosis as well as monitoring treatment outcome, although Hansson et al. (1988) reported little correlation between the MPQ and other pain rating scales (VAS, VDS and NRS) when used to evaluate CDS.

Verbal descriptor checklists

According to Gracely et al. (1978), verbal descriptor checklists appear to allow quantitative assessment of both the sensory and affective dimensions of pain using a continuum across different pain conditions instead of words intended to distinguish conditions (syndromes).

The main disadvantage of rating scales is that pain is assumed to be a unidimensional experience varying only in intensity, and as such a broad range of psychological experience is compressed into an artificially small continuum. Patients tend to spread their responses over the entire scale regardless of the magnitude of the actual sensations (Gracely 1980). Chapman et al. (1985) reported a tendency for investigators to treat scores from studies as interval or ratio level scaling in statistical analysis, without evidence that patients actually use the numbers in this way. Data interpreted in this manner suggest a ranking order and imply that interval differences between the individual values are equal in magnitude, which may not necessarily be true.

Heft & Parker (1984) have shown that category scale values are not equally spaced when labelled with words commonly used to describe pain, and they advocated the use of irregular spacing, which would reflect differences in word meaning.

Price et al. (1983) modified VAS methodology to allow for separate assessment of both intensity and affective (unpleasantness) aspects of pain. Duncan et al. (1989) compared both verbal descriptive checklists and the multidimensional VAS methodology and con-

cluded that both VAS and verbal descriptors successfully quantified sensory intensity and affective aspects of pain, but that verbal descriptors may provide the more sensitive tool for separating intensity and unpleasantness.

The hospital anxiety and depression scale (HAD)

Recently Zakrzewska & Feinmann (1990) employed the hospital anxiety and depression scale (HAD), devised by Zigmond & Snaith (1983), in a 4-year clinical study in patients with atypical facial pain and trigeminal neuralgia, and concluded that the HAD scale was effective in assessing the effect of the reported pain on the wellbeing of the patient. The HAD scale does not appear to have been reported in CDS studies.

Few CDS studies have sought to assess pain intensity and unpleasantness in connection with the patient's oral hygiene activities or in relation to suitable stimuli associated with clinical treatment (Clark et al. 1985).

The patient's fear of possible discomfort from the use of a form of stimulus not normally associated with the clinical situation may also upset the reliability of subjective evaluation of the elicited response. Others, however, have concluded that reliance on subjective response alone would have minimal significance in the evaluation of CDS (Green et al. 1977).

Problems still exist because of investigator inability to observe patient response to external stimuli objectively (Dayton et al. 1974). Threshold measurements alone are insufficient because of variability, and because they are expressed in terms of stimulus rather than perception of pain (McGrath 1986). Variability in pain threshold from patient to patient is attributed to such factors as age, sex, cultural background, attention, suggestion, which may be further modified by various psychological variables (Woodrow et al. 1972, Melzack 1973, Gracely et al. 1978).

Most investigations designed to evaluate the efficacy of desensitizing agents in CDS appear to quantify response by means of criteria which may be described as objective with regard to the method *per se*, but in reality are subjective with regard to patient response. To some extent, the evaluation of treatment for CDS is difficult regardless of the methodology employed.

Methods of assessment of dentinal sensitivity

Mechanical (tactile) stimuli

Different methods of applying mechanical stimuli include scratching the dentine surface with a sharp probe (Cohen 1961, Hernandez et al. 1972, Minkov et al. 1975, Zinner et al. 1977, Uchida et al. 1980, Carlo et al. 1982, Manochehr-Pour et al. 1984, Silverman 1985, Person et al. 1989, Guo-Huo & Morimoto 1991), scaling procedures (Fitzgerald 1956, Everett 1964) as well as mechanical pressure stimulators (Smith & Ash 1964, Kanouse & Ash 1969, Dayton et al. 1974, Green et al. 1977, Lutins et al. 1984, McFall & Morgan 1985, Orchardson & Collins 1987, Kleinberg et al. 1990) and more recently the Yeaple probe (Clark et al. (1987), Minkoff & Axelrod 1987, McFall & Hamrick 1987, Silverman et al. 1988, Kern et al. 1989, Phantumvanit et al. 1990, Prapakamol et al. 1991, Sidi et al. 1991, Gillam et al. 1992a, b).

Explorer probe use to evaluate sensitivity has been criticised. A mechanical probe introduces variability in pressure. Ideally, one would require the same tactile pressure to be exerted on all test teeth at all time intervals during a given clinical trial (Clark & Troullos 1990). The use of scaling procedures has also been criticised, being subject to such factors as pressure applied, instrument sharpness and depth of penetration. Ong & Strahan (1989) questioned whether scratching the dentine with an explorer can be considered a natural stimulus for assessment of CDS. Smith & Ash (1964) developed a mechanical stimulator to provide quantitative information on patient response to scratch stimulation of dentine (Kanouse & Ash 1969, Dayton et al. 1974). This device, subsequently modified (Green et al. 1977, Lutins et al. 1984, McFall & Morgan 1985) incorporated a 15-mm stainless steel wire with a tip ground to a fine point and capable of movement across the buccal surface of the sensitive test tooth. The scratching force could be increased by means of a small screw used to move the tip closer to or away from the root surface. The testing procedure involved moving the wire across the exposed root surface, increasing the scratching force, measured in millimetres, until a painful response (threshold value) was elicited. This device has been criticised since the stimulus intensity could not be measured in force

units, and the size of the device limited its access to the labial surface of the anterior teeth. Smith & Ash (1964) and Green et al. (1977) appear to be the only investigators who have attempted to evaluate the exact position of sensitivity on a given tooth surface, by means of an occlusal relocation key on this device.

Orchardson & Collins (1987) developed a mechanical stimulator comprising a chuck mounted on a short metal beam which carried foil strain gauges. A sickle-shaped caries probe was mounted in the chuck at right angles to the strain gauges. The beam carrying the strain gauges was fixed at its end to the inside of a chrome tube which formed the handle of the instrument. The probe tip was held perpendicular to the tooth which was gently scratched, with gradually increasing force, until the patient indicated that the pain threshold had been reached (minimum stimulus to evoke a sensation of pain). The device was attached to a chart recorder to register the applied force in grams. The investigators claimed that the device afforded easy access to most tooth surfaces, with the exception of the distal aspects of second and third molars and the lingual surfaces of mandibular molars. According to Clark & Troullos (1990), this instrument appeared to provide a quantifiable and reproducible method of assessing CDS.

The Yeaple probe is an electronic pressure-sensitive device originally designed to function as a pressure-controlled periodontal probe (Polson et al. 1980). The probe was modified to accept the tine of a dental explorer (Minkoff & Axelrod 1987, McFall & Hamrick 1987, Clark et al. 1987, Kern et al. 1989). The handle of the probe is approximately the size of a fountain pen and is connected by a flexible electrical lead to a control panel. The probe is designed to deliver a pre-set force when the tip is applied perpendicular to the cervical labial surface. This force may be varied by regulating the current by means of a dial to an electromagnet controlling tip position.

Once the pre-set force is reached a red light shows on the control panel and an audible signal is activated. Application of the incremental probe force (in grams) may be varied by the operator, usually in 5 gram steps (Minkoff & Axelrod 1987, Sidi et al. 1991, Gillam et al. 1992a, b), until the patient experiences discomfort. The force setting is noted at this point. If a maximum force of 70 g

is reached without any perceived discomfort, then the tooth is scored as non-sensitive. McFall & Hamrick (1987) applied pressures of 25, 50 and 75 g in sequence rather than in 5 gram increments. Teeth failing to respond at 75 g were considered non-sensitive and scored 0. Clark et al. (1987) quantified pain by determining which pressure range (<20 g, 20–39 g, 40–59 g, 60–75 g) elicited a painful response. These investigators experienced problems in maintaining constant pressure on the curved surface of the cervical portion of the tooth.

The main advantage of the Yeaple probe is that tactile sensitivity can be reported in terms of a quantifiable, reproducible force (Clark & Troullos 1990). The probe tip also affords access to all tooth surfaces. One of the criticisms of the Yeaple probe is that data analysis requires an assumption that responses over 70/75 g do not exist, or that no response is automatically equivalent to 70/75 g. According to Ash (1986), this problem tends to defeat the use of a scaled stimulus (varied stimulus/constant response test).

Kleinberg et al. (1990) reported a hand-held scratch device, which consisted of a torsion gauge and a sharp explorer-like probe. The device was capable of easy movement across a sensitive tooth and had an indicator, displayed by the arm of the explorer tine, that recorded the force of displacement in centi-newtons. The scratch process was repeated with successively greater force until pain was perceived by the patient. The point at which pain was first perceived was considered the pain threshold. If a tooth failed to respond to a force of 80 cN, it was classified as non-sensitive.

Criticisms applicable to the other methods of assessment by tactile stimuli may be relevant. The use of a sharp probe may also scratch the dentine surface. According to Pashely (1990) pressure, even from a gentle force of 5–10 g, is sufficient to overcome the elastic limit of dentine, leading not only to compression and smear layer creation under the explorer tip, but also to permanent (microscopic) deformation of dentine (scratch development). This deformation of dentine may cause displacement of tubular fluid inwardly at a rapid rate, which activates mechanoreceptors, thereby triggering a pain impulse.

The scratching of the dentine may also remove a therapeutic agent de-

posited during a clinical trial, but this does not seem to substantially influence pain threshold (Smith & Ash 1964).

One of the problems in assessing sensitivity by a scratch test is that the investigator may repeatedly miss the exact location of the sensitive site, leading to a false assumption of non-sensitivity. Several investigators have attempted to identify areas of sensitivity in both *in vivo* and *in vitro* studies (Linden 1968, Ishikawa 1969, Matsumoto et al. 1980, Absi et al. 1987, 1989, Yoshiyama et al. 1989, 1990, Matsumoto et al. 1990, Cuenin et al. 1991, Oyama & Matsumoto 1991).

Chemical (Osmotic) stimuli

Hypertonic solutions, for example, sodium chloride, glucose, sucrose and calcium chloride, have been used to elicit dentinal sensitivity (Anderson & Matthews 1966, Miller et al. 1969, Dayton et al. 1974, Clark et al. 1987, McFall & Hamrick 1987, Ong & Strahan 1989, Prapakamol et al. 1991).

Miller et al. (1969) applied a sugared oral rinse, consisting of sweetened frozen lemon juice concentrate. Although no relevant details were published by the investigators, one may speculate whether the pH of the lemon juice influenced sensitivity by removing the smear layer. Such an effect of pH on (cat) dentine has also been described by Orchardson (1978) and Panopoulos et al. (1983).

Hypertonic solutions have been preferred to acid solutions which have a low pH and as such cause tubular demineralization, which could in turn aggravate sensitivity. Horiuchi & Matthews (1973) demonstrated that hypertonic solutions of sodium chloride, glucose and sucrose elicit pain *in vivo* and also produce fluid movement through dentine *in vitro*. They further reported that hydrostatic pressures were more effective than osmotic pressures in producing fluid shifts. Calcium chloride has multiple effects due to its high solubility. Superficially, it can excite intradental nerves due to osmotic movements (Panopoulos et al. 1983), whereas at deeper levels it may excite nerve activity due to the direct effect of calcium on stabilisation of membranes (Bilotto et al. 1988, Markowitz et al. 1991, Orchardson 1978, 1985).

A warm saturated sucrose solution has been utilised by several investigators (Clark et al. 1987, McFall & Hamrick

1987, Ong & Strahan 1989) as a chemical stimulus. The solution was applied with a cotton bud to the exposed dentine surface for 10 s, or until discomfort was perceived by the patient. Applications of hypertonic solutions to exposed dentine may exert an osmotic effect causing fluid outflow and subsequent pain. Hypertonic solutions of low osmolarity, such as dentinal fluid, will, therefore, have a tendency to flow towards solutions of hyperosmolarity, whereas iso-osmotic solutions when applied elicit no response (Pashley 1986). Panopoulos et al. (1983) demonstrated that while exposed dentine was not, strictly speaking, a semi-permeable membrane, nevertheless the movement of tubular fluid was virtually instantaneous. Horiuchi & Matthews (1973) observed that fluid movements could not always be predicted on the basis of osmotic pressures alone. Johnson & Brännström (1974) concluded that the osmotic properties of a solution were of minor importance with regard to its pain producing effect.

Pashley & Parsons (1987) reported that lidocaine ointment when applied to the gingivae of teeth with exposed dentine elicited pain, possibly as a result of the high polyethylene glycol concentration of the ointment. They postulated that hypertonic solutions, even if they contain local anaesthetic, elicit a pain response if the solution osmotically induces fluid movement through the dentine. The rate of diffusion of the anaesthetic molecules is slower (minutes) relative to the rate of osmotic fluid shift (seconds); hence pain is felt before anaesthesia is obtained.

Anderson et al. (1962, 1966, 1967, 1970) believed that hypertonic solutions were convenient quantifiable stimuli, since chemical concentration could be controlled and osmotic pressure calculated. The efficacy of chemical stimuli, however, may also be influenced by other variables, such as ionic composition, presence or absence of calcium, sodium or potassium, pH and osmolarity (tonicity) (Pashley 1986). Närhi et al. (1988) reported that nerve responses to hypertonic stimulation of superficial dentine were related to the osmotic pressure of the solution used. Hypertonic solutions are generally inconvenient to use and difficult to administer in a controlled manner, and may injure the adjacent soft tissues. Contamination of the tooth may also occur when hypertonic solutions are used as pain stimuli,

which may, in turn, directly increase sensitivity beyond pre-test levels (Pashley 1984). Clark et al. (1987), however, reported no corroborative evidence to support this statement. Chemical stimuli have also been found to be unsuitable for measurement of threshold sensitivity. Anderson et al. (1967) reported that repeated application of hypertonic solutions to prepared cavities in teeth reduced the sensitivity of the surface. There appear to be no studies where the pain threshold has been objectively determined by chemical stimuli.

According to Pashley (1990), Anderson and co-workers in their earlier studies were unaware of the presence and importance of the smear layer, and this, together with the low hydraulic conductance of dentine, necessitated using very large osmotic stimuli to induce sufficient fluid movement through dentine to elicit pain. Johnson & Brännström (1974) reported that a dentine surface covered with a smear layer was much less responsive to hypertonic solutions. Acid etching, for example 50% citric acid for two minutes, will reduce this layer, and consequently the hydraulic conductance of the dentine will be greatly increased (Pashley et al. 1981). The removal of the smear layer will, therefore, enable increased fluid flow through dentine which in turn will increase sensitivity.

This review would, therefore, suggest that recorded responses to hypertonic solutions were neither reliable, predictable nor reproducible, and as such these solutions should not be used as quantifiable stimuli in the assessment of CDS.

Thermal stimuli

Sensitivity to thermal stimuli, especially to cold, appears to be the most prevalent presenting feature in patients complaining of CDS (Harris & Curtin 1976, Kanapka & Colucci 1986, Addy et al. 1987, Orchardson & Collins 1987).

Cold air blast

A 1-s blast of cold air from a dental air syringe has been utilised in the assessment of CDS (Fitzgerald 1956, Levin et al. 1973, Tarbet et al. 1979, 1980, 1982, Uchida et al. 1980, Gangarosa 1981, Carlo et al. 1982, Manochehr-Pour et al. 1984, Silverman 1985, Clark et al. 1987, McFall & Hamrick 1987, Minkoff & Axelrod 1987, Ong & Strahan

1989, Person et al. 1989, Kern et al. 1989, Sidi et al. 1991, Gillam et al. 1992a, b). Cold air blasts, however, may be more useful for identifying individual sensitive teeth during screening rather than a sensitive site, since a cold air blast from a dental air syringe does not help to localise sensitive dentine (Pashley 1990). Ong & Strahan (1989) attempted to remedy this problem by using ribbon wax to isolate the sensitive dentine.

Prolonged air blasts have an unknown and possibly varying temperature effect which can be avoided by using a short application time, typically 1 s (Pashley 1990).

Clark & Troullos (1990) expressed concern that the range of temperature reported led to crossing back and forth over the threshold for each patient. Air blasts, however, cannot be considered graded. They are used as a constant stimulus while the investigator attempts to measure variable patient response (Pashley 1990). It is questionable whether in the absence of a stimulus of graded intensity a change in the threshold of pain can be determined.

Thrash et al. (1983) developed an electronic threshold measurement device which they claimed detected changes in sensitivity and provided a greater degree of objectivity in measuring response to a cold stimulus. This device consisted of a miniature thermistor connected to a chart recorder with an attached hand-held control for patient response. The thermistor was placed adjacent to the sensitive area for an accurate temperature measurement of the point at which the patient first reported pain. Room temperature air (approximately 20°C) was gently blown over a sensitive site (32–34°C), until the patient registered a sensitivity threshold. Measurement of this drop in temperature was repeated 3 × and the average calculated. Some time, however, may be required for the test tooth to return to normal and adaptation to temperature changes may also occur (Kleinberg et al. 1990). For this reason, it is advisable that if both tactile and thermal stimuli are to be used in the same subject, the tactile stimulus should be applied before the thermal stimulus. It is also questionable whether the pain elicited in response to thermal stimuli during this procedure was due solely to cold, as the air jet would also cause dehydration (Ong & Strahan 1989).

A Yeh air thermal system was used

by Minkoff & Axelrod (1987) and Silverman et al. (1988). A temperature controlled stream of air at 10 p.s.i. was directed onto the exposed dentine via a disposable plastic tip. The initial air temperature of 100°F was progressively lowered until a positive response was elicited from the patient or until the lower limit of 70°F was obtained. The air temperature was controlled by passing air from a compressor through copper coiled tubing submerged in an ice bath, to an electrical heating cartridge in the instrument's handle, where the air could be adjusted by a control device. The air temperature was continually monitored by a probe prior to exiting through the instrument tip. This technique appears to be both quantifiable and reproducible, but since the moisture content of the air jet is not controlled, it may have the disadvantage of drying and sensitizing a test tooth as the investigator proceeds down a temperature range (Clark & Troullos 1990, Kleinberg et al. 1990).

Orchardson & Collins (1987) developed an air jet stimulator similar to the system of Thrash et al. (1983), in which the pain threshold was defined by the temperature at the tooth surface. The investigators claimed that it was possible to combine surface temperature with the latency measurements to provide additional information from the same testing procedure with the aid of a small thermistor or thermocouple. The air stimulator developed a controlled jet of air ($20^{\circ}\text{C} \pm 1^{\circ}\text{C}$) from a compressed air supply. The flow of air was regulated by a flow meter which allowed air to pass into a solenoid valve, where it could either be diverted onto the tooth surface (active state) via a nozzle mounted on a transparent perspex carrier, or pass into the room air (inactive state). The device was activated when the operator pressed a foot switch which simultaneously diverted air flow to the tooth and started a three decade digital clock. When the subject experienced a barely perceptible feeling of discomfort he activated a hand held cut-off switch which automatically stopped the clock. The sensitivity was assessed by measuring the time taken for the thermal stimulus to evoke a positive response. Pain threshold was, therefore, expressed as a pain reaction time which was inversely proportional to the sensitivity.

Renton-Harper & Midda (1992) reported an air-jet stimulator (hypersensi-

tivity tester machine) based on that described by Orchardson & Collins (1987).

Recently, a new microprocessor temperature-controlled air delivery system has been developed for determining cold and warm temperature thresholds of dentinal sensitivity, and used in 2 clinical studies (Person et al. 1989). This device consists of a hand held air delivery wand attached to a microprocessor-operated control unit capable of providing a temperature range of -5°C to $+85^{\circ}\text{C}$ ($\pm 0.2^{\circ}\text{C}$). Air is derived from a compressed air source and the flow regulated by a valve to maintain a constant 60 p.s.i. input to the instrument. On entering the instrument, air is delivered tangentially to a vortex separator tube within the wand, where, as a result of the tube design, 2 distinct thermal air streams are produced. In consequence, the air emerging from the front (delivery) end of the tube is cold (-5°C), whereas the air emerging from the rear of the tube is warmer. The cold air stream enters an electrical resistance heater within the air wand and then passes into a standard dental air syringe nozzle. The temperature of the emergent air is monitored by a thermocouple within the nozzle tip which relays this information to the microprocessor control unit. The electrical heater effects rapid and reproducible warming of the emergent airstream. A soft silicone rubber sleeve fits over the air delivery nozzle and allows placement of the nozzle against the tooth surface without discomfort to the patient, and without triggering mechanical stimuli of sensitive dentine surfaces. The air-temperature settings can be adjusted in 1°C increments or decrements using the appropriate buttons on the hand held wand or in 5°C steps by successive depressions of the buttons. Conversely, the desired temperature settings can be entered on the control unit keypad.

From the available data, it would appear that the initial air temperature setting was 39°C , which was gradually lowered in 2 or 1°C decrements until a cold air threshold was reached when the patient perceived discomfort and raised his hand. Following an unspecified period of recovery, the threshold was reappraised for confirmation. The patients were recalled 7 days later, the study teeth re-evaluated and the threshold temperature values from both visits then compared. Of the 236 teeth tested, 113 (47.9%) had identical cold threshold temperatures at both visits: 31 (13.1%)

had differences between $+1^{\circ}\text{C}$ to 5°C , 15 (6.4%) of between $+6$ to 10°C and 77 (32.6%) had differences greater than $+10^{\circ}\text{C}$. The greater cold threshold variability $> +6^{\circ}\text{C}$ observed in 39% of teeth was attributable, according to the investigators, to inherent subject variability in sensitivity perception rather than to instrument error. For warm/hot air thresholds as recorded in study 2 (Person et al. 1989), the initial air temperature setting was at 37°C and increased in 1 or 2°C increments until a warm/hot air threshold was reached in the manner previously described for cold air threshold measurement. This technique would appear to be both quantifiable and reproducible, but the absence of any information relating to the period of recovery between each threshold evaluation and confirmation gives rise to some concern. The problem of simultaneous drying and sensitizing a test tooth as the investigator proceeds down the temperature range has been discussed previously.

The use of prolonged evaporative stimuli has been criticised (Pashley 1990). Brännström (1960) demonstrated that if human dentine was dried with a stream of air for 5 min, it remained insensitive to painful stimuli, as long as it was kept dry. Furthermore evaporative water loss from the dentine caused displacement of odontoblast nuclei into the tubules, although it would appear that desensitization was due to the resultant mechanical blockage (partial tubule occlusion) by the salts and organic substances left behind (Polhagen & Brännström 1971, Pashley et al. 1984).

In summary, the question as to whether the use of air blast stimulation can be refined to the point of providing a quantifiable method of evaluating CDS has yet to be resolved (Pashley 1990).

Cold water testing

Several investigators (Cohen 1961, Miller et al. 1969, Levin et al. 1973) have applied cold water to exposed cervical dentine. Minkov et al. (1975) applied cold water (7°C) from a syringe, while Uchida et al. (1980) utilised 20°C cold water. Flynn et al. (1985) used 15 ml of cold water (7°C) which was rinsed around the mouth for a few seconds. These investigators suggested that cold water at 7°C was ideal for the identification of sensitive teeth as well as minimizing the incidence of false positive

responses. Sensitivity for reasons other than CDS, however, cannot be ruled out.

Cold water testing has also been developed to enable application of water at different temperatures to exposed cervical dentine (Johnson et al. 1982, Brough et al. 1985, Muzzin & Johnson 1989). The thermal testing technique developed by Brough et al. (1985) was modified by Muzzin & Johnson (1989) to include water at temperatures between 20°C and 0°C. The technique involved the use of disposable syringes filled with water from thermally insulated containers at 20, 15, 10, 5 and 0°C. Commencing at 20°C ($\pm 1^\circ\text{C}$), the investigators flowed water over the exposed dentine until a positive response was noted or for a maximum of 3 seconds. If there was no response, the investigators waited 2 min and then retested the tooth with water at 15°C ($\pm 1^\circ\text{C}$). The water temperature was decreased by 5°C decrements until a positive response by the patient was obtained or until the test system limit (0°C $\pm 1^\circ\text{C}$) was reached. The temperature at which a positive response was obtained or, conversely, the lack of response was recorded for each tooth tested. This method is, effectively, a threshold measurement technique.

Cold water testing, however, has been criticized for its lack of objectivity (Green et al. 1977). It is also difficult to determine how much water has been placed on the tooth and the timing of this placement (Gangarosa 1986). It is also difficult to control the flow of water and confine it to a specific tooth or to a specific sensitivity locus. Furthermore, the intensity of the pain perceived by the patient at the temperature which first produced a positive response was not evaluated (Clark & Troullos 1990).

Muzzin & Johnson (1989) stated that they delayed reapplication of water for 2 min between each application of the 5 water temperatures in order to allow the tooth to attain body temperature. It is questionable, however, whether waiting two minutes is sufficient: up to one hour may be required before the tooth can be properly retested again by such means (Jyväsjärvi & Kniffki 1987).

Thermo-electric devices

Quantified thermal (heat and cold) stimuli have been used to determine pre- and post-treatment sensitivity levels. A thermo-electric stimulator (Naylor

1961), modified by Smith & Ash (1964) has been used to report quantitative patient responses to hot and cold (Smith & Ash 1964, Kanouse & Ash 1969, Dayton et al. 1974, Green et al. 1977, Addy et al. 1987). It provided a continuous application of heat or cold via a probe tip small enough to allow placement on the cervical area of the tooth. The temperature of the probe tip was measured with a thermistor embedded in the tip, which enabled the current flow to cool the tip from room temperature to 12°C or conversely to heat it up to 82°C. The initial temperature for thermal sensitivity testing was set at 37.5°C. For cold stimulation, the temperature was reduced in approximately 1°C decrements. At each decrement, the instrument was switched off and the stimulator tip placed in contact with the exposed root surface. This was continued until a positive response was obtained. The procedure for testing the response to heat stimuli was performed in the same manner, except that the temperature of the stimulating tip was increased from the initial temperature of 37.5°C in 1°C increments until a positive response was noted.

McFall & Morgan (1985) used a FTS Direct-Contact-Probe and measuring unit (Model DCP-80, FTS Systems Inc., Stone Ridge, N.Y.), previously used by Lutins et al. (1984) to measure thermal sensitivity, which was capable of providing a temperature range from -80°C to +130°C ($\pm 0.5^\circ\text{C}$). The initial temperature for testing was set at 36°C and lowered by means of an adjustable dial in 1°C decrements. At each decrement, the tip was removed from contact with the tooth for 45 s, and the temperature dial adjusted prior to replacing the tip on the exposed dentine. The procedure was repeated until a positive patient response was noted. The temperature at which this occurred was recorded as the threshold temperature.

Addy et al. (1987), using a similar thermoelectric device to that developed by Naylor (1961), tested for response to cold stimuli by cooling the probe tip to 0°C. Teeth which gave a positive response were then restimulated with the probe set at 5°C. The procedure was then repeated at 10°C and 15°C. The sequential testing of teeth at each temperature allowed an approximately 5-min time interval before the tooth was retested at the next temperature setting. It would appear that no tooth responded at the 10 or 15°C temperature

settings. Ong & Strahan (1989) used a thermal probe unit developed by E. H. Davies (Institute of Dental Surgery, London) which consisted of a thermistor at the probe tip and which housed a water cooled frigistor. The thermal probe was connected by a flexible lead to circuits for temperature measurement, temperature control and constant voltage supply units. The thermistor was capable of providing a temperature range of -5°C to +55°C ($\pm 0.2^\circ\text{C}$), and the device was designed to provide a suitable temperature range for eliciting a sensitivity response to thermal stimuli, via a tip (1.5 mm² surface area of contact) suitable for placement on cervical dentine without contacting the gingival tissue. The investigators appears to test for thermal sensitivity by utilising the extremes of the temperature range, and recorded a response following initial placement of the probe tip for up to a maximum of 10 s. If no possible response was elicited, the probe tip was reapplied after waiting 2 min. Ong (1983) also suggested that thermal testing could be initiated at 37°C, which would give a baseline temperature threshold, and the temperature subsequently adjusted in 1°C increments or decrements until a positive response was recorded.

Thermocouple devices appear to have the advantage of precise control of temperature and to provide accurate threshold values, but unfortunately considerable time is required to set the necessary range of temperatures (Green et al. 1977). These devices register the temperature of the probe tip and not directly that at the tooth surface, and as such suffer from a lag between probe and tooth surface temperatures. Consequently, changes in temperature must be made slowly in order that a temperature threshold of sensitivity is not bypassed (Clark & Troullos 1990). There may also be a problem with placement of a metal tip, even at body temperature, on the exposed dentine, which may trigger a painful response and consequently preclude further testing. Furthermore the heat transfer between a metal probe tip and the tooth depends on a contact area. Problems may also arise with inadequate probe contact (Person et al. 1989) which can result in the presentation to the tooth of poorly characterised and quantified stimuli. Criticism that these devices may not be representative of the real life clinical situation has also been made (Clark & Troullos

1990). Patients who experience CDS normally complain of cold air or cold liquids and not cold solid objects.

Most of the thermal devices presently available require contact with the tooth surface in order to elicit a response, which means that the stimulus is both tactile (mechanical) and thermal in nature. The degree to which thermal stimuli may be considered to be mechanical in nature has yet to be resolved (Ash 1986).

Application of a water stream, however, may be considered to be almost thermal in nature as there is no pressure application. The use of a thermally adjusted airstream provides a no touch thermal stimulation, but unfortunately, as previously discussed, it provides both thermal and evaporative stimuli simultaneously.

According to Pashley (1990), thermal stimuli should be regarded as hydrodynamic in that they induce fluid movement or pressure changes indirectly rather than by directly stimulating temperature-sensitive receptors.

Electrical stimulation

Electrical stimuli have been used by several investigators to quantify both pre-pain and pain thresholds in CDS (Stark et al. 1977, Tarbet et al. 1979, 1980, 1982, Kleinberg et al. 1990). Unlike the other stimuli used to quantify CDS, dentinal tubule fluid movement is not necessary for transmission of the electrical stimulus, but rather the presence of lower resistance organic material in cementum, enamel or dentine (Kleinberg et al. 1990). Electrical stimuli, would, therefore, appear to be more suitable for measuring pulpal activity than for quantifying CDS (Clark & Troullos 1990).

Electrical pulp testers have been utilised to evaluate the vitality of the pulp but the validity of such pulp testing has been called into question (Seltzer & Bender 1975). Furthermore no correlation has been found between pain perception threshold and the histological status of the pulp (Seltzer et al. 1963). Current leakage via the periodontal ligament and subsequent stimulation of periodontal nerves may also yield false positive data. A conventional pulp tester is battery powered, producing pulses of direct current. The intensity of the output voltage (stimulus intensity) may be increased by pre-setting various numbered gradations (0–10) on a thumb wheel. Problems, however, arise in the

interpretation of the information gathered in such a procedure, since it is incorrect to assume a direct relationship between stimulus intensity in volts and the number on the thumb wheel (Kanapka & Colucci 1986). Results from initial studies by these latter investigators clearly demonstrated that conventional pulp testers were not suitable for quantifying CDS.

Stark et al. (1977) developed a dental pulp stethoscope, designed to provide a range of sensitivity levels, which would aid further development of an accurate pulp testing method. The instrument consisted of a digital readout sensitive voltmeter connected to a digital printer apparatus which was activated by a push button control. A conventional battery powered electric pulp tester (Digilog) was attached to the voltmeter. The stimulus intensity was measured in volts (root-mean-square). The pulp tester tip was placed on the mid-gingival third of enamel and the tooth stimulated. A conductive gel with a pH of 5.4–5.6 was used (Ash 1986). On perceiving a tingling or warm sensation, the patient activated a hand-held point control switch which automatically stopped the stimulus and activated the recorder, which printed the voltage needed to produce a current flow that elicited the threshold stimulation. Tarbet et al. (1979, 1980, 1982) suggested there were differences in the electrical pre-pain thresholds in teeth classified as sensitive (using cold air blast) compared to non-sensitive teeth. Similar results were demonstrated by Kleinberg et al. (1990) using a modified Stark instrument, in that sensitive teeth showed both lower pre-pain and pain thresholds than healthy non-sensitive teeth. Stark & Pelgner (1982) suggested that a value of 15 volts and above indicated a range of tooth non-sensitivity. The Stark instrument was evaluated by Tarbet et al. (1979) in a well-controlled double-blind parallel study. The results were comparable to those obtained with the cold air blast stimulus. These investigators reported that electrical stimulation of teeth constituted an accurate and objective method for eliciting and quantifying CDS. The electrical stimulus procedure had the added advantage over the cold air blast in that the threshold stimulus could be approached slowly, so that there would be little associated discomfort.

The methodology of determining threshold values employed by Stark et

al. (1977) and Tarbet et al. (1979), however, has been criticised. One of the problems of electric pulp testing is the risk of the stimulus spreading to adjacent tissues (Orchardson & Collins 1987). To circumvent this, the investigators placed the probe tip on enamel rather than on the sensitive cervical dentine and as such failed to reflect a true dentinal sensitivity. There was also the distinct possibility that by placing the probe tip on enamel the pulpal nerves were directly stimulated rather than the pulp/dentine complex, through indirect stimulation via hydrodynamic forces (Pashley 1990, Clark & Troullos 1990).

Although Tarbet et al. (1979) claimed that their methodology was objective, the patient was able to switch off the stimulus when discomfort was perceived and methodology employed in this study, may, therefore, not be as objective as claimed.

The use of electrical stimuli to quantify CDS has been criticised on the basis of being non-physiology, since the response to such stimuli fails to correspond to the painful response normally experienced by CDS patients. Pashley (1990), however, has suggested that it is theoretically possible for electrical stimuli to induce hydrodynamic fluid movement through open tubules via a phenomenon called electro-osmosis. Pashley concluded that in the absence of current knowledge about this phenomenon (in dentine), electrical stimulation should not be dismissed as non-physiological.

Unlike thermal stimuli, electrical stimuli are not normally encountered in real life situations, and as such there is a question as to the relationship between the voltage values obtained with the electrical stimulus procedure and the pain scale values obtained with normally experienced stimuli.

Fear of experiencing an unknown stimulus and possible discomfort, may, therefore, influence the patient's assessment of pain and in consequence a lower pain threshold value may be recorded. Further, stimulation of the pulp on the basis of applied voltage may fail to represent exact pain threshold values, in as much as the stimulating current depends on varying resistance pathways to the pulp or to other adjacent tissues (Ash 1986). The use of constant current stimulators, as in neurophysiology, capable of delivering an exact current flow regardless of the resistance of the hard tissues of the tooth, has been advocated (Ash 1986, Pashley 1990). Furthermore,

because current flow is the critical variable in stimulating nerves, Pashley (1990) considered the use of constant current stimulators essential in the study of nerve threshold and sensitivity, although ideal stimulators of this type do not appear to be available at this time.

Application of Test Stimuli

The mode and sequence of applying a stimulus which can be varied in intensity is important. Ash (1986) suggested that an increase or decrease in the level of heat or increase in the level of electrical energy should be monotonic rather than delivered in a random order approach. He concluded that while a continuous increase may not be possible, both incremental as well as continuous increases or decreases in stimulus intensity should occur within a standard time frame.

The order of application when more than one kind of stimulus is used is important. Care should be taken to ensure that the 1st should not distract from the 2nd, nor the 2nd from the 3rd and so on. The least disturbing stimulus should, therefore, be applied first, with the most disturbing used last (Ash 1986, Clark & Troullos 1990). Several investigators have applied either tactile, electrical or heat stimuli prior to the application of cold air on the basis that the former do not appear to elicit a painful response which could affect the latter (Tarbet et al. 1979, 1980, 1982, Minkoff & Axelrod 1987, Orchardson & Collins 1987, Addy et al. 1987, Kerns et al. 1989, Person et al. 1989, Gillam et al. 1992a, b). The applied stimulus must be reproducible and behaviour predictable. Without such quantification, it is difficult if not impossible to compare the findings of different investigators (Ash 1986). No method of evaluation, however, may be considered reliable when used alone (Addy & Dowell 1983, Ong & Strahan 1989). There is plainly a need to investigate the measureability and reproducibility of these stimuli using methodologies and instrumentation more related to the clinical situation.

Summary

This review of the literature indicates that there are problems in evaluating patient subjective response to the various stimuli used in the assessment and treatment of CDS. Opinions vary as to the reliability of some of these methods of assessment (Green et al. 1977,

Addy & Dowell 1983, Lecointre et al. 1986, Addy et al. 1987), although more recently efforts have been made to develop controlled reproducible stimuli more suited to the evaluation of CDS (Silverman 1985, Minkoff & Axelrod 1987, McFall & Hamrick 1987, Addy et al. 1987, Clark et al. 1987, Ong & Strahan 1989, Kern et al. 1989, Person et al. 1989, Sidi et al. 1991, Gillam et al. 1992a, b).

Currently, no single method of eliciting and assessing CDS may be considered ideal. The absence of suitably objective methodology of assessing CDS and the lack of standardised measurement of the subjective response following application of stimuli still gives cause for concern.

Further research, is therefore, required to evaluate suitable methodology for the quantification of realistic test stimuli under controlled clinical conditions, whereby the subjective response may be objectively measured.

Zusammenfassung

Beurteilung der Schmerzempfindung bei Studien über zervikale Dentinempfindlichkeit. Eine Übersicht

Diese Übersicht über das Schrifttum läßt erkennen, daß die Auswertung der subjektiven Reaktion des Patienten auf die verschiedenen Stimuli, die zur Beurteilung und Behandlung der CDS (cervical dentine sensibility; zervikale Dentinempfindlichkeit) angewendet werden, problematisch sein kann. Die Auffassungen über die Verlässlichkeit einiger dieser Methoden gehen auseinander, obwohl man sich kürzlich bemüht hat, kontrollierte, wiederholbare Stimuli zu entwickeln, die sich besser zur Beurteilung der CDS eignen. Zur Zeit kann keine einzige Methode zur Stimulation der CDS und seiner Beurteilung als ideal bezeichnet werden. Das Fehlen einer geeigneten objektiven Beurteilungsmethode der CDS und der Mangel an standardisierten Meßverfahren der subjektiven Reaktion auf die Applikation solcher Stimuli, ist beunruhigend. Weiterführende Forschung ist erforderlich, um unter kontrollierten klinischen Bedingungen eine geeignete Methode zur Quantifizierung wirklichkeitsnaher Teststimuli zu finden, die die objektive Messung der subjektiven Reaktion erlaubt.

Résumé

Evaluation de la douleur dans des études de sensibilité dentinaire cervicale. Une revue
Cette revue de la littérature indique qu'il est difficile d'évaluer la réponse subjective du patient à différents stimuli utilisés pour évaluer et traiter la sensibilité dentinaire cervicale. Les opinions varient en ce qui concerne la

fiabilité de certaines méthodes d'évaluation bien que récemment des efforts aient été faits pour développer des stimuli reproductibles et contrôlés, mieux à même d'évaluer la sensibilité dentinaire. Cependant, aucune méthode actuelle de provocation et d'évaluation de la sensibilité dentinaire ne peut être considérée comme idéale. L'absence de méthode objective adéquate permettant d'évaluer cette sensibilité et le manque de mesures standard de la réponse subjective suivant l'application des stimuli, posent toujours un problème. C'est pourquoi, de nouvelles recherches sont nécessaires pour évaluer une méthode adéquate permettant de quantifier les stimuli dans des conditions cliniques contrôlées où la réponse subjective serait mesurée objectivement.

References

- Absi, E. G., Addy, M. & Adams, D. (1987) Dentine hypersensitivity: A study of the patency of dentinal tubules in sensitive and non sensitive cervical dentine. *Journal of Clinical Periodontology* **14**, 280-284.
- Absi, E. G., Addy, M. & Adams, D. (1989) Dentine hypersensitivity: The development of a replica technique to study sensitive and non-sensitive cervical dentine. *Journal of Clinical Periodontology* **16**, 190-195.
- Addy, M. & Dowell, P. (1983) Dentine hypersensitivity - A review. Clinical and in vitro evaluation of treatment agents. *Journal of Clinical Periodontology* **10**, 351-363.
- Addy, M., Mostafa, P., Absi, E. G. & Adams, D. (1985) Cervical dentin hypersensitivity. Etiology and management with particular reference to dentifrices. In: Rowe, N. H. (ed): *Proceedings of Symposium on Hypersensitive dentin. Origin and Management*, University of Michigan, pp 43-64.
- Addy, M., Mostafa, P. & Newcombe, R. (1987) Dentine hypersensitivity. A comparison of five toothpastes used during a 6-week treatment period. *British Dental Journal* **163**, 45-51.
- Anderson, D. J., Hannam, A. G. & Matthews, B. (1970) Sensory mechanisms in mammalian teeth and their supporting structures. *Physiology Rev* **50**, 171-195.
- Anderson, D. J. & Matthews, B. (1966) An investigation into the reputed desensitizing effect of applying silver nitrate and strontium chloride to human dentine. *Archives of Oral Biology* **11**, 1129-1135.
- Anderson, D. J. & Matthews, B. (1967) Osmotic stimulation of human dentine and the distribution of dental pain thresholds. *Archives of Oral Biology* **12**, 417-426.
- Anderson, D. J., Matthews, B. & Gorretta, C. (1967) Fluid flow through human dentine. *Archives of Oral Biology* **12**, 209-216.
- Anderson, D. J., Matthews, B. & Shelton, L. E. (1967) Variations in the sensitivity of osmotic stimulation of human dentine. *Archives of Oral Biology* **12**, 43-47.
- Anderson, D. J. & Ronning, G. A. (1962) Osmotic excitants of pain in human dentine. *Archives of Oral Biology* **7**, 513-523.

- Ash, M. M. (1986) Quantification of stimuli. *Endodontology & Dental Traumatology* **2**, 153–156.
- Bilotto, G., Markowitz, K. & Kim, S. (1988) Effects of ionic and non-ionic solutions on intradental nerve activity. *Pain* **32**, 231–238.
- Brännström, M. (1960) Dentinal and pulpal response – 2. Application of an air stream to exposed dentine. An experimental study. *Acta Odontologica Scandinavica* **18**, 17–28.
- Brough, K. M., Anderson, D. M., Love, J. & Overman, P. R. (1985) The effectiveness of iontophoresis in reducing dentin hypersensitivity. *Journal of the American Dental Association* **111**, 761–765.
- Carlo, G. T., Ciancio, S. G. & Seyrek, S. K. (1982) An evaluation of iontophoretic application of fluoride for tooth desensitization. *Journal of the American Dental Association* **105**, 452–454.
- Chapman, C. R., Casey, K. L., Dubner, R., Foley, K. M., Gracely, R. H. & Reading, A. E. (1985) Pain measurement. An overview. *Pain* **22**, 1–31.
- Clark, D. C., Al-Joburi, W. & Chan, E. C. S. (1987) The efficacy of a new dentifrice in treating dentin sensitivity. Effects of sodium citrate and sodium fluoride as active ingredients. *Journal of Periodontal Research* **22**, 89–93.
- Clark, G. E. & Troullos, E. S. (1990) Designing hypersensitivity clinical studies. *Dental Clinics of North America* **34**, 531–544.
- Cohen, A. (1961) Preliminary study of the effects of a strontium chloride dentifrice for the control of hypersensitive teeth. *Oral Surgery, Oral Medicine, Oral Pathology* **14**, 1046–1052.
- Council on Dental Therapeutics. (1986) ADA Ad hoc Advisory Committee: Recommendations for evaluating agents for the reduction of dentinal hypersensitivity. *Journal of the American Dental Association* **112**, 709–710.
- Cuenin, M. F., Scheidt, M. J., O'Neal, R. B., Strong, S. L., Pashley, D. H., Horner, J. A. & Van Dyke, T. E. (1991) An *in vivo* study of dentin sensitivity. The relation of dentin sensitivity and the patency of dentin tubules. *Journal of Periodontology* **62**, 668–673.
- Dayton, R. E., DeMarco, T. J. & Swedlow, D. (1974) Treatment of hypersensitive root surfaces with dental adhesive materials. *Journal of Periodontology* **45**, 873–878.
- Downie, W. W., Leatham, P. A., Rhind, V. M., Wright, V., Branco, J. A. & Anderson, J. A. (1978) Studies with pain rating scales. *Annals of the Rheumatic Diseases* **37**, 378–381.
- Duncan, G. H., Bushnell, M. C. & Lavigne, G. J. (1989) Comparison of verbal and visual analogue scales for measuring the intensity and unpleasantness of experimental pain. *Pain* **37**, 295–303.
- Ekowski, C., Hrubes, V., Joyce, C. R. B., Flood, M. K., Mason, R. M. & Zutshi, D. W. (1972) An experimental study of two methodological problems in clinical evaluation. Different types of scales and the availability of patients' previous judgments. *Psychopharmacology* (suppl.) **26**, 70.
- Everett, F. G. (1964) Desensitization of hypersensitive exposed root surfaces. *Dental Clinics of North America* (March), pp. 221–230.
- Fitzgerald, G. (1956) A clinical evaluation of a new agent for the relief of hypersensitive dentine. *Dental Digest* **62**, 494–497.
- Flynn, J., Galloway, R. & Orchardson, R. (1985) The incidence of 'hypersensitive' teeth in the West of Scotland. *Journal of Dentistry* **13**, 230–236.
- Gangarosa, L. P. (1981) Iontophoresis application of fluoride by tray techniques for desensitization of multiple teeth. *Journal of the American Dental Association* **102**, 50–52.
- Gangarosa, L. P. (1986) Letters to the Editor. *Journal of the American Dental Association* **112**, 808–810.
- Gillam, D. G., Newman, H. N., Bulman, J. S. & Davies, E. H. (1992^a) Dentifrice abrasivity and cervical dentinal hypersensitivity. Results 12 weeks following cessation of 8 weeks' supervised use. *Journal of Periodontology* **63**, 7–12.
- Gillam, D. G., Newman, H. N., Davies, E. H. & Bulman, J. S. (1992^b) Clinical efficacy of a low abrasive dentifrice for the relief of cervical dentinal hypersensitivity. *Journal of Clinical Periodontology* **19**, 197–201.
- Gracely, R. H. (1980) Pain measurement in man. In: Ng, L. K. Y. & Bonica, J. J. (eds.): *Pain discomfort and humanitarian care*. New York, Elsevier North Holland, pp. 111–138.
- Gracely, R. H., McGrath, P. & Dubner, R. (1978) Ratio scales of sensory and affective verbal pain descriptors. *Pain* **5**, 5–18.
- Green, B. L., Green, M. L. & McFall, W. T. (1977) Calcium hydroxide and potassium nitrate as desensitizing agents for hypersensitive root surfaces. *Journal of Periodontology* **48**, 667–672.
- Guo-Hua, L. & Morimoto, M. (1991) Magnesium sulphate as a desensitizing agent. *Journal of Oral Rehabilitation* **18**, 363–372.
- Hall, E. H., Terezhalmay, G. T. & Pelleu, G. B. (1986) A set of descriptors for the diagnosis of dental pain syndromes. *Oral Surgery, Oral Medicine, Oral Pathology* **61**, 153–157.
- Hansson, R. E., Bye, F. L. & Smith, B. A. (1988) Four different Pain Rating Scales Used to Evaluate Dentin Hypersensitivity. *Journal of Dental Research* **67** (spec. issue), 1433 (abstr.).
- Harris, R. & Curtin, J. H. (1976) Dentine hypersensitivity. *Australian Dental Journal* **21**, 165–169.
- Heft, M. W. & Parker, S. R. (1984) An experimental basis for revising the category rating scale for pain. *Pain* **19**, 153–161.
- Hernandez, F., Mohammed, C., Shannon, I., Volpe, A., King, W. (1972) Clinical study evaluating the desensitizing effect and duration of two commercially available dentifrices. *Journal of Periodontology* **43**, 367–372.
- Horiuchi, H. & Matthews, B. (1973) In vitro observations on fluid flow through human dentine caused by pain producing stimuli. *Archives of Oral Biology* **18**, 275–294.
- Huskišson, E. C. (1974) Measurement of pain. *Lancet* **2**, 1127–1131.
- Ishikawa, S. (1969) A clinico-histological study on the hypersensitivity of dentin. *Journal of the Japanese Stomatology Society* **36**, 68–88, 278–298 (In Japanese).
- Johnson, G. & Brännström, M. (1974) The sensitivity of dentine – Changes in relation to conditions at exposed tubule apertures. *Acta Odontologica Scandinavica* **32**, 29–38.
- Johnson, R. H., Zulqar-Nain, B. J. & Koval, J. J. (1982) The effectiveness of an electroionizing toothbrush in the control of dentinal hypersensitivity. *Journal of Periodontology* **53**, 353–359.
- Joyce, C. R. B., Zutshi, D. W., Hrubes, V. & Mason, R. M. (1975) Comparison of fixed interval and visual analogue scales for rating chronic pain. *European Journal of Clinical Pharmacology* **8**, 415–420.
- Jyväsjärvi, E. & Kniffki, K. D. (1987) Cold Stimulation of Teeth: A Comparison between the responses of cat intradental A-d and C fibres and human sensation. *Journal of Physiology* **391**, 193–207.
- Kanapka, J. A. (1990) Over-the-Counter dentifrices in the treatment of tooth hypersensitivity. Review of clinical studies. *Dental Clinics of North America* **34**, 545–560.
- Kanapka, J. A. & Colucci, S. V. (1986) Clinical evaluation of dentinal hypersensitivity. A comparison of methods. *Endodontics & Dental Traumatology* **2**, 157–164.
- Kanouse, M. C. & Ash, M. M. (1969) The effectiveness of a sodium monofluorophosphate dentifrice on dentinal hypersensitivity. *Journal of Periodontology* **40**, 38–40.
- Keele, K. D. (1948) The pain chart. *Lancet* **11**, 6–8.
- Kern, D. A., McQuade, M. J., Scheidt, M. J., Hanson, B. & Van Dyke, T. E. (1989) Effectiveness of sodium fluoride on tooth hypersensitivity with or without iontophoresis. *Journal of Periodontology* **60**, 386–389.
- Kleinberg, I., Kaufman, H. W. & Confessore, F. (1990) Methods of measuring tooth hypersensitivity. *Dental Clinics of North America* **34**, 515–529.
- Lecointre, C., Apiou, J., Marty, P. & Poitou, P. (1986) Controlled trial of the action of a toothpaste containing nicomethanol hydrofluoride in the treatment of dentine hypersensitivity. *Journal of International Medicine Research* **14**, 217–222.
- Levin, M. P., Yearwood, L. L., Carpenter, W. N. (1973) The desensitizing effect of calcium hydroxide and magnesium hydroxide on hypersensitive dentin. *Oral Surgery, Oral Medicine, Oral Pathology* **35**, 741–746.
- Linden, L. A. (1968) Microscopic observations of fluid flow through cementum

- and dentine. *Odontologisk Revy* **19**, 367–381.
- Lutins, N. D., Greco, G. W. & McFall, W. T. (1984) Effectiveness of sodium fluoride on tooth hypersensitivity with and without iontophoresis. *Journal of Periodontology* **55**, 285–288.
- Manochehr-P, M., Bhat, M. & Bissada, N. (1984) Clinical evaluation of two potassium nitrate toothpastes for the treatment of dental hypersensitivity. *Periodontal Case Reports* **6**, 25–30.
- Matsumoto, K., Izumi, M. & Nagasawa, H. (1980) Scanning electron microscopic study on the hypersensitivity of dentin. *Japanese Journal of Conservative Dentistry* **23**, 247–251.
- Matsumoto, K., Oyama, T., Shirasuka, T. & Wakabayashi, H. (1990) Study on the replica of dentin tubules by silicon-resin method. *Journal of Showa University of Dentistry* **10**, 203–205.
- McFall, W. T. & Hamrick, S. W. (1987) Clinical effectiveness of a dentifrice containing fluoride and a citrate buffer system for the treatment of dentinal sensitivity. *Journal of Periodontology* **58**, 701–705.
- McFall, W. T. & Morgan, W. C. (1985) Effectiveness of a dentifrice containing formalin and sodium monofluorophosphate on dental hypersensitivity. *Journal of Periodontology* **56**, 288–292.
- McGrath, P. A. (1986) The measurement of human pain. *Endodontics & Dental Traumatology* **2**, 124–129.
- Melzack, R. (1973) *The puzzle of pain*. Basic Books, New York.
- Melzack, R. (1975) The McGill Pain Questionnaire: major properties and scoring methods. *Pain* **1**, 277–299.
- Melzack, R. & Wall, P. (1988) *The challenge of pain*. Penguin Books, pp. 34–46.
- Miller, J. T., Shannon, I. L., Kilgore, W. G. & Bookman, J. E. (1969) Use of a water-free stannous fluoride-containing gel in the control of dental hypersensitivity. *Journal of Periodontology* **40**, 490–491.
- Minkoff, S. & Axelrod, S. (1987) Efficacy of strontium chloride in dental hypersensitivity. *Journal of Periodontology* **58**, 470–474.
- Minkov, B., Marmari, I., Gedalia, I. & Garfunkel, A. (1975) The effectiveness of sodium fluoride treatment with or without iontophoresis on the reduction of hypersensitive dentin. *Journal of Periodontology* **46**, 246–249.
- Mumford, J. M. (1973) *Toothache and related pain*. Edinburgh & London, Churchill Livingstone, pp. 57–107.
- Muzzin, K. B. & Johnson, R. (1989) Effects of potassium oxalate on dentin hypersensitivity in vivo. *Journal of Periodontology* **60**, 151–158.
- Närhi, M., Ngassaa, D., Hirvonen, T. & Karhunen, V. (1988) The threshold of dog intradental nerves to osmotic stimulation of dentine. *Journal of Dental Research* **172**, (abstr.).
- Naylor, M. N. (1961) A thermo-electric tooth stimulator. *British Dental Journal* **110**, 228–230.
- Ohnhaus, E. E. & Adler, R. (1975) Methodological problems in the measurement of pain. A comparison between the verbal rating scale and the visual analogue scale. *Pain* **1**, 379–384.
- Ong, G. (1983) *Clinical trial of a desensitising dentifrice*. M.Sc. Thesis, University of London, pp. 42–49.
- Ong, G. & Strahan, J. D. (1989) Effects of a desensitizing dentifrice on dentinal hypersensitivity. *Endodontics and Dental Traumatology* **5**, 213–218.
- Orchardson, R. (1978) The generation of nerve impulses in mammalian axons by changing the concentration of the normal constituents of extracellular fluid. *Journal of Physiology* **275**, 177–189.
- Orchardson, R. (1985) Is calcium more effective than strontium as a desensitizing agent for dentine? In: Lisney, S. J. W. & Matthews, B. (eds.): *Current topics in oral biology*. Bristol, University of Bristol press, pp. 205–215.
- Oyama, T. & Matsumoto, K. (1991) A clinical and morphological study of cervical hypersensitivity. *Journal of Endodontics* **17**, 500–502.
- Panopoulos, P., Gazelius, B. & Olgart, L. (1983) Response of feline intradental sensory nerves to hyperosmotic stimulation of dentin. *Acta Odontologica Scandinavica* **41**, 369–375.
- Pashley, D. H. (1984) Smear layer: physiological considerations. *Operative Dentistry* (suppl. 3), 13–29.
- Pashley, D. H. (1986) Sensitivity of dentin to chemical stimuli. *Endodontics & Dental Traumatology* **2**, 130–137.
- Pashley, D. H. (1990) Mechanisms of dentin sensitivity. *Dental Clinics of North America* **34**, 449–473.
- Pashley, D. H., Kehl, T., Pashley, E. & Palmer, P. (1981) Comparison of in vitro and in vivo dog dentin permeability. *Journal of Dental Research* **60**, 763–768.
- Pashley, D. H., O'Meara, J. A., Kepler, E. E., Galloway, S. E., Thompson, S. M. & Stewart, F. P. (1984) Dentin permeability. Effects of desensitizing dentifrices in vitro. *Journal of Periodontology* **55**, 522–525.
- Pashley, D. H. & Parsons, G. S. (1987) Pain produced by topical anaesthetic ointment. *Endodontics & Dental Traumatology* **3**, 80–82.
- Pashley, D. H., Galloway, S. E. & Stewart, F. P. (1984) Effect of fibronogen in vivo on dentine permeability in dogs. *Archives of Oral Biology* **29**, 725–728.
- Person, P., Demand, E. E., Koltun, L. & Spindel, M. (1989) A microprocessor temperature-controlled air delivery system for dentinal hypersensitivity testing. *Clinical Preventive Dentistry* **11**, 3–9.
- Phantumvanit, P., Prapakamol, S., Angchanpen, O. & Songpaisan, P. (1990) Clinical study of Aluminum Lactate in desensitizing dentifrices. *Journal of Dental Research* **69**, 1977 (abstr.).
- Polhagen, L. & Brännström, M. (1971) The liquid movement in desiccated and rehydrated dentin in vitro. *Acta Odontologica Scandinavica* **29**, 95–102.
- Polson, A. M., Caton, J. G., Yeaple, R. N. & Lander, H. A. (1980) Histological determination of probe tip penetration into gingival sulcus of human using an electronic pressure sensitive probe. *Journal of Clinical Periodontology* **7**, 479–488.
- Prapakamol, S., Angchanpen, O., Songpaisan, Y. & Phantumvanit, P. (1991) Al-Lactate desensitizing dentifrice by mechanical, thermal and chemical stimuli. *Journal of Dental Research* **70**, 4 (abstr.).
- Price, D. D., McGrath, P. A., Raffi, A. & Buckingham, B. (1983) The validation of visual analogue scales as ratio measurements of experimental and chronic pain. *Pain* **17**, 45–56.
- Renton-Harper, P. & Midda, M. (1992) NdYAG laser treatment of dentinal hypersensitivity. *British Dental Journal* **172**, 13–16.
- Scott, J. & Huskisson, E. C. (1976) Graphic representation of pain. *Pain* **2**, 175–184.
- Seltzer, S. & Bender, I. B. (1975) The nerve supply of the pulp and pain perception. In: *The dental pulp*, 2nd edition. Philadelphia, J. B. Lippincott Company, pp. 131–151.
- Seltzer, S., Bender, I. B. & Ziontz, M. (1963) The diagnosis of pulp inflammation, correlations between diagnosis data and actual histological findings in the pulp. *Oral Surgery, Oral Medicine, Oral Pathology* **16**, 846–871.
- Seymour, R. A. (1982) The use of pain scales in assessing the efficacy of analgesics in post-op dental pain. *European Journal of Clinical Pharmacology* **23**, 441–444.
- Sidi, A. D., Wilson, R. F. & Ashley, F. P. (1991) Efficacy of a new toothpaste formulation in reducing dentine hypersensitivity. *Journal of Dental Research* **70**, 110 (abstr.).
- Silverman, G. (1985) The sensitivity-reducing effect of brushing with a potassium nitrate-sodium monofluorophosphate dentifrice. *Compendium of Continuing Education in Dentistry* **6**, 131–136.
- Silverman, G., Gingold, J. & Clark, G. E. (1988) The effectiveness of KNO₃ and NaMFP dentifrices in reducing dentinal hypersensitivity. *Journal of Dental Research* **67**, 1076 (abstr.).
- Smith, B. A. & Ash, M. M. (1964) Evaluation of a desensitizing dentifrice. *Journal of the American Dental Association* **68**, 639–647.
- Smith, B. A. & Ash, M. M. (1964) A study of a desensitizing dentifrice and cervical hypersensitivity. *Journal of Periodontology* **35**, 222–231.
- Smith, B. A., Hansson, R. E., Caffesse, R. G. & Bye, F. L. (1988) Evaluation of dipotassium oxalate in treatment of root hypersensitivity. *Journal of Dental Research* **67**, 1733 (abstr.).
- Stark, M. M., Kempler, D., Pelzner, R. B., Rosenfeld, J., Leung, R. L. & Mintatos, S. (1977) Rationalization of electric pulp-

- testing methods. *Oral Surgery, Oral Medicine, Oral Pathology* **43**, 598–606.
- Stark, M. M. & Pelgner, R. (1982) Measurement of dentinal hypersensitivity. *Compendium of Continuing Education in Dentistry* (suppl. 3) 110–112.
- Stephen, R. M. (1937) Correlation of clinical tests with microscopic pathology of dental pulp. *Journal of Dental Research* **16**, 267–278.
- Tarbet, W. J., Silverman, G., Stolman, J. M. & Fratarcangelo, P. A. (1979) An evaluation of two methods for the quantification of dentinal hypersensitivity. *Journal of the American Dental Association* **98**, 914–918.
- Tarbet, W. J., Silverman, G., Stolman, J. M. & Fratarcangelo, P. A. (1980) Clinical evaluation of a new treatment for dentinal hypersensitivity. *Journal of Periodontology* **51**, 535–540.
- Tarbet, W. J., Silverman, G., Fratarcangelo, P. A. & Kanapka, J. A. (1982) Home treatment for dentinal hypersensitivity. A comparative study. *Journal of the American Dental Association* **105**, 227–230.
- Thrash, W. J., Dorman, H. L. & Smith, F. D. (1983) A method to measure pain associated with hypersensitive dentin. *Journal of Periodontology* **54**, 160–162.
- Uchida, A., Wakano, Y., Fukuyama, O., Miki, T., Iwayama, Y. & Okada, H. (1980) Controlled clinical evaluation of a 10% strontium chloride dentifrice in treatment of dentin hypersensitivity following periodontal surgery. *Journal of Periodontology* **51**, 578–581.
- Woodrow, K. M., Friedman, G. D., Siegel, A. B. & Collen, M. F. (1972) Pain tolerance: differences according to age, sex and race. *Psychosomatic Medicine* **34**, 548–556.
- Yoshiyama, M., Masada, J., Uchida, A. & Ishida, H. (1989) Scanning electron microscopic characterization of sensitive versus insensitive human radicular dentin. *Journal of Dental Research* **68**, 1498–1502.
- Yoshiyama, M., Noiri, Y., Ozaki, K., Uchida, A., Ishikawa, Y. & Ishida, H. (1990) Transmission electron microscopic characterization of hypersensitive human radicular dentin. *Journal of Dental Research* **69**, 1293–1297.
- Zakrzewska, J. M. & Feinmann, C. (1990) A standard way to measure pain and psychological morbidity in dental practice. *British Dental Journal* **169**, 337–339.
- Zigmond, A. S. & Snaith, R. P. (1983) The Hospital Anxiety and Depression Scale. *Acta Psychologica Scandinavica* **67**, 361–370.
- Zinner, D. D., Duany, L. F. & Lutz, H. J. (1977) A new desensitizing dentifrice. Preliminary report. *Journal of the American Dental Association* **95**, 982–985.

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Development and laboratory evaluation of the Biomat Thermal Probe (BTP). E.H.DAVIES, D.G.GILLAM*, H.N.NEWMAN, J.S.BULMAN & I.MILLAR. (Institute of Dental Surgery, London, U.K.)

Thermo-electric devices have been claimed to provide accurate threshold values when compared with other methods of evaluating cervical dentinal sensitivity (CDS) (Green *et al.*, J Periodontol 48: 667-672, 1977). This study reports the development of a thermo-electric device (Biomat Thermal Probe [BTP]), a pilot model of which has been used in CDS assessment and nerve recovery studies (Ong 1983, Talhi *et al.* J Dent Res 64: 694 (Abstr. 286), 1985). In vitro consistency tests with the BTP consisted of a series of 5 readings at set temperatures from 0-59.9°C. BTP calibration was from a selected ambient temperature of 24°C to 59.9°C, 59.9°C-24°C; 24°C-0°C, 0°C-24°C; and from 24°C to 59.9°C and 0°C in 1° and 5°C increments and decrements respectively. All data were subjected to regression analysis. Analysis between BTP tip and set temperatures gave a correlation coefficient of 0.99. The time for BTP to reach the set temperatures was consistent for all tests. 1°C increments/decrements for both cold and heat were obtained within 1-3 seconds and 5°C increments within 6-30 seconds (time increased as 0-4°C was approached) for cold and 6 seconds for heat respectively. There was a slight lag in the time for set temperatures to be reached as measured at the probe tip. The results demonstrated that the BTP is accurate in in vitro measurement of temperature (range 0°C-59.9°C, with SD 0.2°C for 1°C change in tip temperature). Further in vivo studies are indicated to compare the BTP with recognised methods of assessment of patient subjective response in CDS. This study was supported by Block Drug Co. Inc. NJ, USA.

Quantification of Pain in Cervical Dentinal Sensitivity
(CDS) Studies. D.G.GILLAM* J.S.BULMAN and H.N.NEWMAN.
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Previous studies have utilised verbal and non-verbal scales for subjective assessment of pain following application of tactile, thermal, electrical and chemical stimuli. This study compared continuous VAS, 0-10 numerical rating VAS, separate intensity (IVD) and unpleasantness verbal descriptor (UVD) scales (Duncan et al., Pain 37:295-303, 1989) to quantify sensory and affective aspects of pain. 25 patients (8M+17F) rated their overall assessment of sensitivity to every day stimuli and pain perception following tactile (Yeaple probe) and thermal stimuli (dental air syringe). An unweighted moving average technique was used to construct graphs of the relative frequency of reported severity over a range of 0-10. These indicated that cold air caused the greatest discomfort, tactile the least, with the air intensity curve for both IVD and 0-10 VAS peaking at severity level 5, continuous VAS at level 3-4. All methods peaked at level 2 for tactile sensitivity. The UVD scale peaked at level 2-3 and again at 6 for air sensitivity, but conformed to the other scales by peaking at level 2 for tactile sensitivity. With the exception of the UVD scale, the 0-10 numerical rating VAS and IVD scales provided acceptable alternatives to continuous VAS; indicative of the imprecise nature of the words of the UVD scale. **The results confirm that both continuous and numerical rating VAS, together with IVD scales but not UVD quantify both sensory and affective aspects of pain.** This study was supported by Block Drug Co. Inc. NJ, USA.

Effect of SCH Dentifrices on Plaque and Gingivae.
D.G.GILLAM H.N.NEWMAN* and J.S.BULMAN. Institute of
Dental Surgery, London UK.

It has been suggested (Addy et al. Clin Prev Dent 12:28-33,1990) that a silica-based strontium acetate and fluoride dentifrice was more effective in reducing plaque than a strontium chloride hexahydrate (SCH) dentifrice containing the abrasive diatomaceous earth. This study compared two SCH dentifrices, similar except for abrasive (diatomaceous earth or silica-based) on plaque and gingivae in a 2 month randomised double-blind parallel clinical study involving 40 patients (15M+25F). Plaque and gingivitis were assessed using the indices of Silness & Loe and Loe & Silness respectively. There was a slight non-significant increase in plaque at 2 weeks from baseline, (paired 't' test, 19 d.f.), but negligible change thereafter, the effect being identical in both groups. Mean PlI for both groups at 2,4,8 weeks was 0.64-0.68 (SE \pm 0.040-0.064). Unpaired t-tests of between-group differences at 2,4 and 8 weeks yielded t-values of which the highest was only 0.45 (NS). Similarly there was a slight GI change, slightly higher in the diatomaceous earth group, the highest unpaired t-value (for the difference in scores between baseline and 4 weeks) being only 1.34 (NS). Mean GI for both groups at 2,4,8 weeks was 0.52-0.63 (SE \pm 0.216-0.263). The increase did not extend beyond the boundaries of the 95% confidence intervals for the true mean scores. The results would not support previous findings that SCH increased plaque accumulation. Neither dentifrice had any clinically significant effect on plaque and gingival condition.

**D.G.GILLAM*, J.S.BULMAN & H.N.NEWMAN. Institute of Dental Surgery London, U.K..
Use of Word Descriptors in Cervical Dental Sensitivity (CDS) Studies.**

Questionnaires such as the McGill Pain Questionnaire (MPQ) have been used to evaluate painful conditions including CDS. This study aimed to identify key words associated with CDS using word descriptors from the MPQ. 40 subjects (25F+15M) mean age 42.8 years (SD 8.18) recruited for a 2 month randomised double-blind clinical study (Gillam et al.1992 J.Clin Perio 19:197-201) described their discomfort from CDS by completing a MPQ word descriptor form on 2 separate occasions (0 & 56 days). Words commonly selected were sharp,tender,annoying,stabbing,aching and nagging. A Wilcoxon 2-sample rank test was used to examine the changes in scores between the test (silica-based) and control (diatomaceous-earth) groups,on all 130 scores (Test) and 113 scores (Control),regardless of subject.The result was not significant (SND=1.26). Each subject's contribution to the total number of scores was weighted. The average numbers of scores for both Test and Control were 6.5 and 5.65 respectively (Average 6.1). An SND test of the difference in proportions showing a higher score on the second visit between the 2 groups was not significant (SND=1.39 unweighted,0.59 weighted). Percentage reproducibility 78/217 (36%) & 70/201 (34.8%) respectively for both Test & Control groups does not suggest a relationship between the words selected,nor did comparison of weighted scores over the 2 visits. The results indicate word descriptors may be of limited value in the assessment of CDS. This study was supported by Block Drug Co. Inc, NJ, USA.

D.G. GILLAM*, H.N. NEWMAN, E.H. DAVIES, and J.S. BULMAN. Institute of Dental Surgery, London, U.K): Dentifrice abrasivity and cervical dental hypersensitivity.

To clarify the significance of abrasivity in desensitizing dentifrices, diatomaceous earth and silica-based strontium chloride hexahydrate-containing dentifrices were compared in a 2 month randomised, double-blind parallel cervical dental hypersensitivity (CDH) study and 3 months thereafter. Two CDH groups, 20 subjects each, were evaluated for tactile (Yeaple probe) and air sensitivity (dental air syringe) and subjective perception of pain (Visual Analogue Scale VAS). % reductions in CDH were as follows:- Yeaple probe (force increase) for silica group 22.5 at 8 weeks; 15.4 at 20 weeks; diatomaceous group, 24.6 at 8, 18.2 at 20 weeks; tactile VAS, for silica group 46.3 at 8, 32.8 at 20 weeks; diatomaceous group 48.5 at 8 weeks; 49.3 at 20 weeks. Cold air blast, for silica group, 48.3 at 8, 37.7 at 20 weeks; for diatomaceous group, 44.3 at 8 weeks, 37.0 at 20 weeks; Subjective Overall VAS, for silica group 53.2 at 8 weeks, 31.1 at 20 weeks; for diatomaceous group 51.4 at 8 weeks, 45.3 at 20 weeks. There was no difference between groups at any time ('t' tests). 12/52 after dentifrice use, in spite of slight regression, reductions were still significant relative to baseline (Yeaple and Tactile VAS $p < 0.01$, air $p < 0.001$, overall sensitivity $p < 0.05$ (silica), $p < 0.01$ (diatomaceous) (paired 't' tests). It was concluded that changing abrasive component did not significantly affect desensitization. CDH reductions by both dentifrices were still evident and comparable 3 months after cessation of regular controlled use. This study was supported by Block Drug Co. Inc., U.S.A.

Clinical Evaluation of the Biomat Thermal Probe (BTP)

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Problems in evaluating the clinical effectiveness of desensitizing agents appear to derive from a lack of objective methodology. Hence the variety of methods used to assess cervical dentinal sensitivity (CDS). Opinions vary as to reliability^{1,2}. This study reports the clinical evaluation of a thermo-electric device (BTP), a pilot model of which has been used for the assessment of CDS and in nerve recovery studies^{3,4}. Eleven patients (5F, 6M) mean age 43.8 years (SD 6.58) who provided written informed consent and had at least one tooth sensitive to tactile (probe) and thermal (cold air) stimuli were tested for reproducibility of hot and cold thresholds on two occasions (0 and 7 days). The thermal challenge (BTP) was in 5°C increments/decrements commencing from 25°C, the interstimulus time interval was 1 minute and the stimulus was applied for 10 seconds. A one second blast of cold air (dental unit syringe 19°C-24°C, 40-65 p.s.i.) was used for comparison. Objective response observations were supplemented by patient subjective response utilizing Visual Analogue Scales (VAS) forms. All data were normally distributed and paired t-tests were utilized. No statistically significant differences were demonstrated for BTP cold threshold or VAS scores (26 teeth) for cold (t-test 0.48, (25df), 95% C.I.: -1.63-2.62) and VAS (t-test 0.15 (25df), 95% C.I.: -0.70-0.80) respectively. There were no significant differences between visits for hot threshold values (t-test 0.86 (25df), 95% C.I.: 0.72-1.75) and cold air VAS (cm) scores (t-test 0.16 (25df), 95% C.I.: -0.79-0.67). Percentage reproducibility of BTP cold threshold values (°C) indicated that 19/26 teeth (73.1%) had differences of <5°C and 7/26 teeth (26.9%), had differences >8°C. The results demonstrate that the BTP is both accurate and consistent in the measurement of cold threshold stimulation temperatures and would warrant inclusion in clinical studies designed to evaluate the efficacy of desensitizing agents. This study was supported by Block Drug Co., Inc. New Jersey, USA.

REFERENCES

- Green BL, Green ML and McFall WT. Calcium Hydroxide and potassium nitrate as desensitizing agents for hypersensitive root surfaces. *J Periodontol* 48: 667-672, 1977.
- Addy M and Dowell P. Dentine hypersensitivity - A review. Clinical and *in vitro* evaluation of treatment agents. *J Clin Periodontol* 10: 351-363, 1983.
- Ong G. Clinical trial of a desensitizing dentifrice. MSc Thesis, University of London: 42-49, 1983.
- Talhi FS, Davies EH and Nally FF. Nerve recovery after cryotherapy for paroxysmal trigeminal neuralgia. *J Dent Res* 64: 694, Abstr. No. 286, 1985.

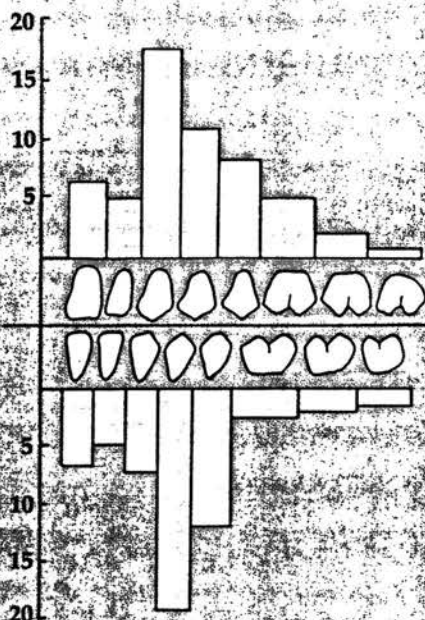


Figure 3: Frequency with which sensitivity was recorded in each tooth of the permanent set (expressed as a percentage)

Continued from p3

suggests that, should the rate of flow of the tissue fluid be increased suddenly, the odontoblast would be sucked against the inner opening of the tubule, like a plug in a sink. This in turn might cause pressure and stimulation of the nerve endings in this area. The application of cold items, sweet solutions or solid objects such as a dental probe, to the outer dentine surface have all been shown to cause a sudden increase in the rate of flow of the fluid. Cold items will contract the fluid, sweet solutions have an osmotic effect and solid objects exert a capillary attraction; all have the effect of sucking more fluid from the tubules. This would explain why all three types of stimuli cause the instant, sharp pain.

Heat, on the other hand, has been shown to cause an initial stasis of the fluid flow followed by a reversal of the direction of flow. In the opinion of many authorities, the clinical observation that heat applied to dentine causes

a duller, more slowly developing pain than the other types of stimuli, confirms the hydrodynamic mechanisms as the means by which stimuli applied to the dentine are conveyed to the pulpal nerves.

Why is exposed dentine not always sensitive?

If the hydrodynamic theory is accepted, pain would be prevented if the dentinal tubules were blocked for any reason. This would prevent any fluid movement in the tubules. On the outer aspect the tubules could be blocked by remnants of cementum, food debris or by tartar accumulating on the root surface. (Some patients complain of transient dentine sensitivity following scaling of the teeth by a dental hygienist or dentist.) It has been shown by electron microscopy that in areas of sensitive dentine there are always at least some of the tubules with patent openings at the outer surface. Obviously this has important

significance in relation to the treatment of the condition.

However, the tubules can also be "blocked" at their inner (pulpal) end. Throughout the life of a tooth, odontoblasts retain the ability to lay down further dentine (secondary dentine). Such dentine is deposited at a very slow rate throughout life and is thought to be the reason that dentine sensitivity is more common amongst young adults than the elderly, who have less sensitive teeth despite gingival recession increasing with age.

If dentine becomes exposed to the oral cavity (either by gingival recession or due to

caries) the secondary dentine is laid down much more rapidly, presumably as a defensive mechanism. This dentine is much more irregular in structure than the original dentine with far fewer tubules. The reduced number of patent tubules tends to reduce sensitivity.

If the hydrodynamic theory is accepted, then the logical means of controlling dentine sensitivity are either:

- block the dentinal tubules, or
- prevent the transmission of pain by the nerves associated with the odontoblasts.

This is discussed in the next section.

Treating sensitivity

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Sensitive teeth are a major cause of mouth discomfort. Sensitivity interferes with our enjoyment of many cold, sweet and sour foods, often to the point of our having to be very careful about what we eat if we want to avoid pain.

It is important to realise that such sensitivity may be the first sign of tooth decay, or even of tooth fracture or a defective filling, and dental advice should be sought in the first instance.

The clue to the condition lies in its technical name, cervical dentinal sensitivity or hypersensitivity.

The principal solid tooth tissue is dentine, or ivory. The dentine forms a tissue complex with the central core of tooth blood vessels and nerves, the dental pulp. Fine tubules extending from the pulp through the dentine to its outer surface, contain cell processes and fluid.

Tiny changes in the hydrodynamic and ion characteristics of this complex structure result in stimulation of the pulp nerve endings which are mainly, if not all, pain nerve endings. Normally these serve as an excellent warning system, but if the outer dentine is not sufficiently protected, then the patient will often experience dentinal sensitivity.

Many factors can cause this exposure. The crown dentine is usually covered by enamel, and the root dentine by cementum, a bone-like tissue. These are insensitive. Often

there is an anatomical gap between the two. Excessive toothbrush abrasion of the overlying cementum (the hardened enamel resists wear) may lay bare the dentine.

Particularly important is the extension of the fine tubules all the way through the dentine with their openings on the outer dentine surface. These may be exposed due to the absence of covering enamel and cementum, to the removal of cementum and also dentine by abrasion, or by acids, for example, from foods or from stomach regurgitation.

Such factors also work against the saliva, which normally replenishes mineral lost from the tooth surface, since saliva is super-saturated with hydroxyapatite, the principal calcium phosphate component of enamel, cementum and dentine.

Treatments are many, and it must be said that to date none have proved to be universally effective.

Most treatments are chemical in type, with an increasing tendency to utilise a number of physical and physico-chemical techniques to treat, or at least to aid in uptake and/or retention of desensitising agents.

Discomfort from cervical dentinal sensitivity (CDS) or dental hyper-sensitivity is a common finding within the adult population. Recent surveys have indicated that one in four adults suffer from this condition. Females appear to suffer more than males, probably due to their overall

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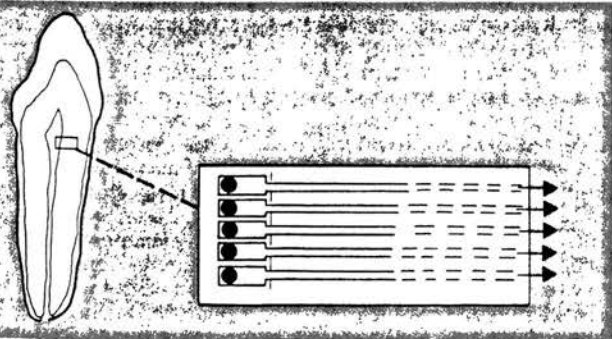


Figure 4: Portion of dentine, illustrating the odontoblasts lining the pulpal surface of the dentine with the odontoblast processes passing into the dentinal tubule. Arrow indicates direction of flow of tissue fluid when the outer surface of the dentine is exposed to the oral cavity

Continued from p5

health care and better oral hygiene awareness. Sensitivity appears to peak in incidence at the end of the third decade and the start of the fourth.

The pain of cervical dentinal sensitivity is rapid in onset, sharp in character and of short duration although, the pain on occasions may persist as a dull or vague sensation in the affected tooth — commonly the eye (canine) and premolar teeth. The surface mostly affected is the one facing the cheeks. Excessive brushing is a probable cause of exposing the underlying dentine surface.

Extremes of temperature appear to trigger sensitivity, cold being the more common complaint according to those suffering from this condition.

Management of patients suffering from CDS should be based on a correct diagnosis of the condition by the dentist who should be able to provide advice on correct brushing procedures so as to minimise further damage to the exposed root surface of the tooth.

One cannot underestimate the importance of counselling about intake (especially frequency) of acidic fruits and beverages in relation to when the teeth are brushed.

Further management should be based on the severity of the condition. For example, for isolated problems, therapy is largely professionally delivered and should be directed towards varnishes, adhesives, filling materials and cervical restorations.

For general sensitivity affecting several teeth, the use of over-the-counter dentifrices may be advised. These contain an active ingredient which claims to reduce cervical dentinal sensitivity by either occluding (blocking) the open tubules at the root surface or by blocking nerve activity.

These therapeutic topically-applied agents may be broadly classified into the following groups based on their supposed mode of action:

- 1) Anti-inflammatory drugs such as steroids (prednisolone)
- 2) Protein precipitants such as formalin, strontium chloride
- 3) Tubule occluding agents including fluoride, potassium nitrate/chloride, potassium oxalate, ferric oxalate and strontium chloride or acetate.
- 4) Tubule sealants: resins or varnishes.
- 5) Miscellaneous: laser, hypnosis, restorative materials, glass ionomer cements.

Of the several mechanisms of desensitisation proposed for desensitising dentifrices, the most widely accepted modes of action are tubule occlusion

and alteration of sensory nerve activity through potassium ion concentration.

Dentifrices containing formalin have in the past been shown not to be effective in reducing sensitivity and such dentifrices do not appear to be available now.

Strontium chloride (in dentifrice form), which appears to have a dual action of protein precipitation and/or tubule occlusion, has been claimed to be effective in reducing sensitivity, although this effect may not last.

The use of fluoride pastes and mouth rinses has also been advocated but, despite their widespread use in most western countries, there does not appear to be a drastic reduction in sensitivity. Fluoride's use as an effective anticaries agent should, however, be encouraged.

Other dentifrices containing potassium nitrate, citrate or chloride, act not through blockage of the open dentinal tubule at the root surface, but by altering the activity of the sensory nerve within the dentine itself. Various laboratory studies have, however, failed to substantiate the claims of these desensitising agents, for example, potassium nitrate and strontium chloride, other mechanisms of action may, therefore be responsible for their reported clinical success.

Recent laboratory studies have highlighted a number of potential agents, such as the oxalates, although their claims, too, need to be substantiated in clinical trials against existing recognised agents.

Alternative forms of treatment, such as hypnosis, have been proposed. Laser technology has been reported to relieve CDS through creating an altered surface layer on the root physically occluding the tubule, although further research is needed before this technique can be recognised as an acceptable treatment and the risks of tooth damage by incorrect use are considerable.

In cases where persistent, long term sensitivity has been a problem, restorative materials such as glass ionomer cements, resins and adhesives have been reported to reduce sensitivity, although once the seal between material and root surface breaks down sensitivity may return.

To date no single desensitising agent or therapeutic technique, despite claims to the contrary, appears to provide a satisfactory long-term solution to this persistent clinical problem, which is currently the subject of much research interest.

PRODUCT APPROVAL

Accreditation schemes for toothpaste

The toothpaste packet without an accreditation logo is rapidly becoming the exception.

However the existence of two separate schemes — British Dental Association accreditation and British Dental Health Foundation accreditation — creates confusion for pharmacists and consumers.

The BDA scheme, launched in 1990, with the first products accredited in September 1991, was the first such scheme and appears to be the more popular of the two with manufacturers. Products and their claims are evaluated by a panel of experts appointed by the BDA, which represents 26,000 dentists.

Accreditation can take a number of months and it is normally granted for a period of three years, and can be renewed if the product continues to meet the BDA requirements. A fee of £20,000 covers the cost of assessing a full brand. Products awarded BDA accreditation are entitled to use the BDA logo.

Quality marks

All the Macleans toothpastes have had BDA approval since August 1991. David Bradley, oral care marketing manager says Smithkline Beecham decided to submit their products for BDA approval because "it was the first scheme and seemed to be the more rigorous scheme."

Stafford Miller favours accreditation schemes that "will help the industry as a whole", and say they submitted Sensodyne F for BDA accreditation because it was the first scheme in operation and was being run by a well-respected and representative body.

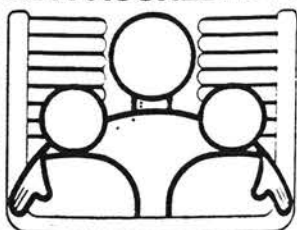
Michael Bealing, chemist development manager at Colgate, was less enthusiastic about the schemes which are "potentially confusing" and "only validate the claims made by manufacturers."

Although Colgate had received BDA approval for Colgate Gum Protection Formula in 1991, the logo is only beginning to appear on packs in response to the actions of other companies.

Crest Tartar Control, Crest Decay Prevention and Crest Ultra Protection also have BDA accreditation.

The British Dental Health Foundation, a registered

BDA ACCREDITED



BRITISH DENTAL ASSOCIATION



charity, aims to promote the benefits of achieving and maintaining the highest standards of dental care to the public. Their accreditation scheme was set up in October 1991 and is intended to cover consumer products in the field of dental health care.

Products with supporting data are submitted fourteen working days before the sitting of the BDHF accreditation panel and the verdict is given on the day. Submitting a product for approval costs £3,500, and if the application is successful the annual retention fee is £5,000 per product.

Market use

The BDHF accreditation logo can be used on product packaging, advertising and promotional material with an accreditation statement.

Mentadent P, SR and Signal toothpaste all have BDHF accreditation. According to Rod Connors, dental product manager for Elida Gibbs, the quality of both accreditation schemes is exactly the same.

Other products that have BDHF accreditation are the range of toothpastes from Boots, Asda, Sainsburys, Gateway, Safeway and Tesco, and Lloyds Dentalcare Sensitive Toothpaste.

Unfortunately, accreditation schemes, introduced in an attempt to differentiate between toothpastes, may have added to the confusion.